

Acta Genetica et Statistica Medica

In association with

Otto L. Mohr

Professor of Anatomy, Oslo

Tage Kemp

Professor of Human Genetics,
Copenhagen

edited by:

Gunnar Dahlberg

Head of the State Institute of Human Genetics and Race Biology, Uppsala

Vol. III

1952

Fasc. 1

INDEX

Juvenile Amaurotic Idiocy. By S. Rayner	1
Methodologische Bedenken zur Zwillingsforschung. Von E. Hug	6
Studies on the Eruption of Permanent Teeth. II. By P. Adler and E. Gödeny	30
Menarche in Schoolgirls. By T. Romanus	50
The Mortality Rate of Cancer Yesterday and Today. By G. Dahlberg	61
A Note on the Dilemma of Human Genetics. By G. Dahlberg	69
The Social Outlook for Children of Divorcees. By E. Otterström	72



BASEL (Switzerland)

S. KARGER

NEW YORK

The „*Acta Genetica et Statistica Medica*“ is issued quarterly. Each issue has approximately 96 pages. The annual subscription rate is Swiss frs. 48.—.

No payment is made for contributions, but 50 reprints of the article will be sent to the author free of charge. Extracopies, if desired, will be supplied at a special rate. The cost of the engravings will be borne by the publishers, provided the figures and graphs are suitable for reproduction and do not exceed a reasonable number. Otherwise the author after due notification, will be charged with the additional cost. Articles will be printed in English, French, and German, with summaries of about 10 lines. As a rule only original papers can be accepted.

All manuscripts should be addressed to *Gunnar Dahlberg*, State Institute of Human Genetics and Race Biology, Uppsala (Sweden). Corrected proofs, review copies, however, as well as enquiries concerning subscriptions and notices, should be sent to the publishers, *S. Karger Ltd.*, Holbeinstrasse 22, Basle (Switzerland).

Les „*Acta Genetica et Statistica Medica*“ paraissent en fascicules trimestriels d'environ 96 pages. Le prix de l'abonnement annuel est de frs. suisses 48.—.

Les collaborateurs reçoivent à titre d'honoraires pour leurs travaux originaux 50 tirages à part gratuits. Les tirages à part supplémentaires seront facturés à un prix modéré. La maison d'édition se charge des frais de clichés à condition qu'elle reçoive des originaux se prêtant à la reproduction et dont le nombre ne dépasse pas la mesure strictement nécessaire. Autrement les frais supplémentaires seront, après avertissement, à la charge de l'auteur. Les travaux pourront être rédigés en langue anglaise, française ou allemande et doivent être suivis d'un court résumé d'environ 10 lignes. Ne seront acceptés en principe que des travaux originaux inédits.

Tous les manuscrits sont à adresser au Prof. Dr. *Gunnar Dahlberg*, State Institute of Human Genetics and Race Biology, Uppsala (Suède). Les épreuves corrigées, les ouvrages à analyser, de même que toute correspondance concernant les abonnements et la publicité sont à adresser à *S. Karger S. A.*, Editeurs, Holbein-strasse 22, Bâle (Suisse).

Die „*Acta Genetica et Statistica Medica*“ erscheinen vierteljährlich in Heften von etwa 96 Seiten zum Jahresabonnementspreis von Schweiz.-Fr. 48.—.

Mitarbeiter erhalten für ihre Originalarbeiten an Stelle eines Honorars 50 Sonderdrucke kostenfrei; weitere Separata gegen mäßige Berechnung. Die Kosten der Clichés übernimmt der Verlag, soweit reproduktionsfähige Vorlagen geliefert werden und die Zahl der Abbildungen das notwendige Maß nicht überschreitet. Andernfalls gehen die Mehrkosten zu Lasten des Autors und werden vorher mitgeteilt. Die Arbeiten können in englischer, französischer oder deutscher Sprache eingereicht werden und sind mit einer kurzen, etwa zehnzeiligen Zusammenfassung zu versehen. Es werden grundsätzlich nur unveröffentlichte Originalarbeiten angenommen.

Alle Manuskripte sind zu richten an Prof. Dr. *Gunnar Dahlberg*, State Institute of Human Genetics and Race Biology, Uppsala (Schweden). Korrigierte Fäden, Rezensionsexemplare sowie Zuschriften, Abonnemente und Inserate betreffend, sind an den Verlag *S. Karger A. G.*, Holbeinstraße 22, Basel (Schweiz) zu senden.

From the Institute of Genetics, University of Lund, Sweden
Department of Medical Genetics (Head at the time: Jan A. Böök¹⁾, M. L., F. L.)

JUVENILE AMAUROTIC IDIOCY²⁾

Diagnosis of heterozygotes

By S. RAYNER

Since the extensive study by *Sjögren* [1931] *juvenile amaurotic idiocy* (*Spielmeyer-Vogt*) has been known as a clinical and genetical entity due to a single recessive gene difference, the diseased individuals being homozygotes. Like other rare recessive genetic diseases *juvenile amaurotic idiocy* appears to occur with a different frequency in different geographical regions (isolates) in Sweden. The average morbidity risk has been estimated at 1: 40,000 live births. From this figure, which also equals the mutation rate of this gene per generation as the condition is semilethal, the incidence of the heterozygotes is calculated at about one half of a per cent. These figures, of course, are correct if the population follows the *Hardy-Weinberg* law, only. As this according to the above-mentioned uneven geographical distribution at least should be doubted, the frequency of heterozygotes in the Swedish population would be somewhat lower (cf. *Dunn* [1947]). Important in this connection, however, is the fact only, that heterozygotes in spite of the rareness of the disease occur in an appreciable number, probably exceeding 1: 1,000.

If we agree to the existence of genes, none of them, more or less arbitrarily classified as dominants or recessives, could be entirely without effect. In medical genetics it remains an important task to try to detect the effect of so-called recessive genes in single dose. A diagnosis of heterozygotes for harmful recessive genes would open the field for many important studies. As an example one could mention

¹⁾ At present Assistant Director at the State Institute of Human Genetics, Uppsala, Sweden.

²⁾ Aided by a grant from Reservationsanslaget för Främjandet av Medicinsk Forskning.

the importance of studying the fertility of heterozygotes who marry genetically normal partners, a question of crucial significance for the genetic understanding of populations especially when mutation rates are calculated. From a practical point of view the importance of being able to diagnose such heterozygotes for purposes of genetic counseling is obvious. It should perhaps be added that one does not necessarily have to assume that the effect of such recessive genes in *single* dose is harmful and when such an effect has been demonstrated the gene is no longer recessive but intermediate in action. A number of problems related to this topic, together with a list of genetic diseases which more or less accurately can be diagnosed in heterozygous condition were discussed recently by Neel [1949].

In 1948 *Bagh* and *Hortling* reported the discovery of a vacuolization of the lymphocytes in six cases of *juvenile amaurotic idiocy* in Finland. Simple blood smears stained according to the common *May-Grünwald-Giemsa* scheme were used. Rather large clear vacuoles were observed in the cytoplasma in a varying percentage of the lymphocytes. The results of these two authors have been summarized in table 1. With the exception of this peculiar vacuolization of the lymphocytes blood cytology appeared normal. The nature of these vacuoles is still unknown. *Bagh* and *Hortling* recommend their finding as an additional diagnostic aid.

The present author has undertaken (1) a recheck of *Bagh* and *Hortling's* results and (2) a pilot test to find out if this vacuolization of the lymphocytes might occur in the heterozygotes (i.e. the parents of patients with *juvenile amaurotic idiocy*).

Though the data are still very limited the results appear consistent enough to justify a report. As the disease is rare it would be highly desirable if the findings be rechecked and controlled where material is available.

The technic used was the same as that of *Bagh* and *Hortling* [1948], i.e. the common routine staining with the *May-Grünwald-Giemsa* solution. In every case 100–300 lymphocytes were studied.

The results of seven examined cases of typical *juvenile amaurotic idiocy*, all inmates of The Institution for the Blind with Complicating Defects at Lund, Sweden, has been summarized in table 2. The existence of vacuolized lymphocytes was substantiated in every case (cf. fig. 1), although the percentage of abnormal cells was in most cases lower than reported by *Bagh* and *Hortling*.

So far the parents of cases no. 11 and 13 (sibs) and of cases no. 39 and 41 (also sibs) have been examined. All four parents showed vacuolized lymphocytes (cf. fig. 2) but were otherwise healthy as judged from a routine physical examination.

1) for the parents of sib-pair 11-13, the mother 10 per cent vacuolized lymphocytes and the father 11 per cent.

2) for the parents of sib-pair 39-41, the mother 14 per cent vacuolized lymphocytes and the father 4 per cent.

Furthermore the sibs of the index cases 11 and 13 have been traced. Another case of *juvenile amaurotic idiocy* in this sibship was deceased. Of eight living sibs seven were examined. Six siblings had vacuolized lymphocytes, varying from 3 to 9 per cent. One sister (no. 7), as well as four spouses of the sibs did not show any vacuolization. The available data have been summarized in the pedigree (fig. 3).

Table 1. Vacuolization of lymphocytes in Juvenile Amaurotic Idiocy.
Data by Bagh and Hortling [1948].

Case no.	Age in years	Duration of the disease in years	Percentage vacuolized lymphocytes
1	17	10	56
2	14	7	51
5	12	5	36
6	10	4	31
3	11	2.5	22
4	7	1	12

Table 2. Vacuolization of lymphocytes in Juvenile Amaurotic Idiocy.
Present data.

Case no.	Age in years	Duration of the disease in years	Percentage vacuolized lymphocytes
56	20	14	14
11	20	13	8
13	15	10	5
39	18	10	17
69	13	7	19
70	12	6	12
41	12	5	22

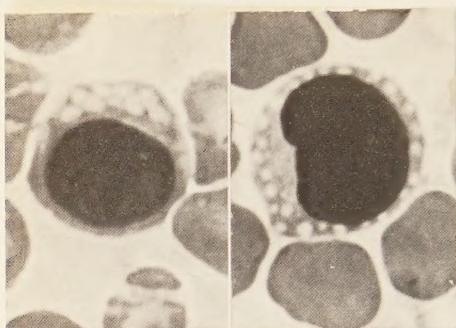


Fig. 1

Fig. 2

Fig. 1. Vacuolized lymphocyte of case of *Juvenile Amaurotic Idiocy (homozygote)*. Case no. 39. 1700 \times . *Fig. 2.* Vacuolized lymphocyte of heterozygote for *Juvenile Amaurotic Idiocy*. Normal Father of case no. 11. 1700 \times .

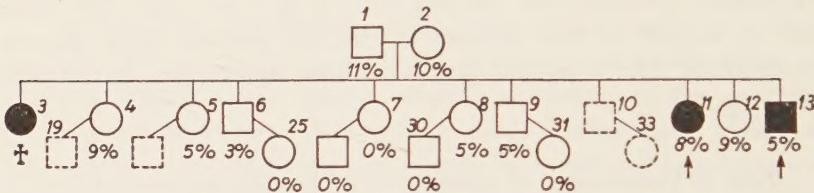


Fig. 3. A family with 3 cases of *Juvenile Amaurotic Idiocy* (black individuals) where both parents and several siblings display vacuolization of lymphocytes (percentage of vacuolized cells noted below the square or circle). Arrows point to propositi. Not examined individuals in broken lines. The figures above the symbols give the number of the individuals.

Summary.

Vacuolized lymphocytes as reported by *Bagh* and *Hortling* [1948] in *juvenile amaurotic idiocy* would appear to be a consistent sign and was found in 7 Swedish cases. The same type of vacuolization furthermore was seen in four parents of two unrelated cases and in six out of seven normal siblings of one family. Four normal spouses of these siblings had a normal blood cytology. Though the data are very limited and conclusions must be guarded the findings indicate (1) that the vacuolization is correlated with the disease and very likely an effect of the same genes and (2) that the vacuolization found in the normal relatives (parents and some sibs) might be an expression of the gene for *juvenile amaurotic idiocy* in single dose. The possibility of diagnosing the heterozygotes for *juvenile amaurotic idiocy* by the simple test of looking for vacuolized lymphocytes will be explored in further pedigrees.

Résumé.

Comme il fut communiqué par *Bagh* et *Hortling* [1948] des lymphocytes vacuolisés semblent être un phénomène habituel dans

l'idiotie amaurotique juvénile; l'auteur en a trouvés chez 7 cas suédois. Une telle vacuolisation pouvait en outre être constatée chez les 4 parents de 2 cas différents et chez 6 de 7 frères et sœurs normaux d'une même famille. 4 époux normaux de ces frères et sœurs avaient une cytologie du sang normale. Les données étant si limitées, il faut qu'en se garde de conclusions. Pourtant les trouvailles indiquent (1) que la vacuolisation est liée à la maladie et probablement conditionnée par les mêmes gènes; (2) que la vacuolisation trouvée chez des parents et quelques frères et sœurs pourrait être l'effet d'un seul des gènes conditionnant l'idiotie amaurotique juvénile en dose double. La possibilité de diagnostiquer les hétérozygotes pour la maladie en cherchant des lymphocytes vacuolisés sera étudiée dans d'autres arbres généalogiques.

Zusammenfassung.

Vakuolierte Lymphozyten, wie sie von *Bagh* und *Hortling* [1948] bei juveniler amaurotischer Idiotie beschrieben werden, scheinen ein sicheres Kennzeichen zu sein und wurden in 7 schwedischen Fällen gefunden. Derselbe Typ von Vakuolisation wurde ferner bei vier Eltern zweier nichtverwandter Fälle und bei sechs von sieben normalen Geschwistern einer Familie festgestellt. Vier normale Gatten dieser Geschwister hatten eine normale Blutzytologie. Obgleich die Daten sehr begrenzt sind und Schlußsätze genau überprüft werden müssen, zeigen die Befunde auf: 1. daß zwischen der Vakuolisation und der Krankheit eine Korrelation besteht und sehr wahrscheinlich eine Einwirkung von denselben Genen sind, und 2. daß die Vakuolisation, welche bei den normalen Verwandten (Eltern und einige Geschwister) gefunden wurde, ein Ausdruck der Gene für juvenile amaurotische Idiotie in einzelner Dosis sein könnte. Die Möglichkeit der Diagnose der Heterozygoten für juvenile amaurotische Idiotie durch die einfache Probe der Nachschau nach vakuolisierten Lymphozyten soll an weiteren Stammbäumen erforscht werden.

Acknowledgement. The author wishes to express his gratitude to Dr. J. A. Böök for advice and help during the course of this investigation.

BIBLIOGRAPHY

- Bagh, K. V. and H. Hortling:* Blodfynd vid juvenil amaurotisk idioti. Nord. med. 38, 1072–76, 1948. – *Dunn, L. C.:* The effects of isolates on the frequency of a rare human gene. Proc. Acad. Sc. 33, 12, 359–363, 1947. – *Neel, J. V.:* The detection of genetic carriers of hereditary diseases. Am. J. Human Genetics 1, 19–36, 1949. – *Sjögren, T.:* Die juvenile amaurotische Idiotie. Hereditas 14, 197–425, 1931.

METHODOLOGISCHE BEDENKEN ZUR ZWILLINGSFORSCHUNG

Von ERIK HUG, St. Gallen

Die folgenden Darlegungen sind aus einem Vortrag hervorgegangen, den ich kürzlich in der Naturforschenden Gesellschaft St. Gallen hielt. Sie bezwecken keineswegs eine vollständige Kritik der Zwillingsforschung als Methode der menschlichen Erblehre. Diese ist einer späteren Arbeit vorbehalten. Ich greife hier nur einige wenige Gesichtspunkte heraus, von denen aus sich die Zwillingsmethode kritisch beleuchten lässt. Das wichtige Gebiet der Manifestationswahrscheinlichkeit ist z. B. nicht behandelt. Selbst die zentrale Frage der Zwillingsforschung, das Erbe-Umweltproblem, konnte nur gestreift werden. Als vorläufige Mitteilung mögen die folgenden Ausführungen jedoch genügen. Es handelt sich im wesentlichen um eine Zusammenfassung der häufigst gemachten Einwände, wobei die Akzente da und dort etwas anders gesetzt und die Konsequenzen schärfster gezogen werden, als das bisher geschah. Wer sich über die ziemlich umfangreiche Methodenkritik näher orientieren will, sei vor allem auf die Arbeiten von Dahlberg (1926–50), Lenz (1932–48) und Gottschick (1937) verwiesen.

Die Methode.

Die Zwillingsforschung, wie sie heute als Methode der menschlichen Erblehre betrieben wird, geht in ihren Grundlagen auf *Francis Galton*, den Begründer der Eugenik, zurück. Schon der Titel seiner Abhandlung – „The history of twins as a criterion of the relative powers of nature and nurture“ – lässt erkennen, was *Galton* bereits 1876 mit der Gegenüberstellung von identischen und nichtidentischen Zwillingen¹ bezweckte: er glaubte auf die Weise den Einfluß von

¹ identische = erbgleiche oder eineiige Zwillinge, nichtidentische = erbverschiedene oder zweieiige Zwillinge. Die synonymen Ausdrücke identisch und nichtidentisch sind von *Galton* zweifellos im Sinne von erbgleich und erbverschieden gemeint (Lenz 1950).

Erbe (nature) und Umwelt (nurture) am Zustandekommen eines Merkmals *getrennt* bestimmen zu können. „Die Lebensgeschichte der Zwillinge gestattet uns nämlich, die Wirkung jener Kräfte, die ihnen von Geburt an die Richtung weisen, von der Wirkung jener Kräfte zu trennen, welchen sie erst durch die Umstände des späteren Lebens ausgesetzt sind“. Die Macht der Vererbung lasse sich so sinnfällig demonstrieren, besonders bei solchen identischen Zwillingen, die trotz verschiedener Umweltbedingungen, denen sie während ihrer Entwicklungszeit ausgesetzt waren, in der Regel keine großen Merkmalsdifferenzen zeigten, während nichtidentische Zwillinge trotz gleicher Umwelt starke Unterschiede aufwiesen.

Diese Pionierarbeit aus vormendelistischer Zeit ist lange unbeachtet geblieben, da sich eine fruchtbare Auswertung des ihr zugrundeliegenden Gedankens erst durchführen ließ, als man Mittel und Wege fand, die eineiigen Zwillinge (EZ) von den zweieiigen (ZZ) mit genügender Sicherheit zu trennen. Es geschieht dies heute mit Hilfe der sogenannten polysymptomatischen Ähnlichkeitsdiagnose, die *Siemens* 1924 in die Zwillingspathologie einführte. *Siemens* ging von der Überlegung aus, daß sich EZ und ZZ auch ohne bekannten Eihautbefund bestimmen lassen, wenn sie sich in einer größeren Anzahl erblicher Merkmale unterscheiden, d. h. wenn sich verschiedene genetisch bedingte Eigenschaften bei eineiigen Zwillingen vorwiegend konkordant (merkmalsähnlich), bei zweieiigen Zwillingen vorwiegend diskordant verhalten. Damit stand der Erbforschung am Menschen ein fast unbeschränktes Material zur Verfügung, was in der Folge zu einem ungeahnten Aufschwung der Zwillingsforschung führen sollte. Von 1924 an datiert also diese zur Zeit häufigst angewandte Methode der Humangenetik, die zwar nicht den Erbgang eines Merkmals, wohl aber den relativen Anteil von Erb- und Umweltbeeinflußbarkeit desselben bestimmen will, wie dies schon *Galton* versuchte. Darüber hinaus soll die Größe der Modifikationsbreite bzw. der Grad der Manifestationsschwankung einer Eigenschaft festgestellt werden.

Siemens möchte die Zwillingsforschung in Parallele zu den Versuchen mit reinen Linien stellen. Denn ein EZ-Paar ist einer reinen Linie vergleichbar, bestehend aus zwei genotypisch identischen Individuen, deren Merkmalsunterschiede ausschließlich paratypisch (neben- oder umweltgeändert) bedingt sind, während die Merkmalsunterschiede der zweieiigen Zwillinge auch von der verschiedenen Erbanlage der Paarlinge abhängen. Durch Vergleich der Ähnlich-

keitsbefunde bei den eineiigen Zwillingen erhält man Einblick in die Paravariabilität oder Modifizierbarkeit einer Eigenschaft und kann so die Vorfrage jeder Erbuntersuchung entscheiden. Entsprechendes gilt für die sogenannte Manifestationswahrscheinlichkeit eines Merkmals. Die Hauptfrage der Zwillingsforschung ist jedoch diejenige nach dem *Grad der Erblichkeit* einer Eigenschaft, zu welchem Zwecke man das betreffende Merkmal an einer Reihe von eineiigen und zweieiigen Paaren untersucht. Ein Leiden z. B., das vorwiegend erblich bedingt ist, muß bei EZ häufiger konkordant angetroffen werden als bei ZZ. Umgekehrt ist jedes Leiden vorwiegend nichterblicher Natur, wenn es bei EZ und ZZ etwa in gleicher Übereinstimmung festgestellt wird. Aus dem jeweils vorliegenden Konkordanzverhältnis läßt sich also die verschiedengradige Erb- und Umweltbedingtheit aller jener Merkmale ablesen, die zwischen den beiden Polen „rein erbbedingt“ und „rein umweltbedingt“ liegen.

Das ist in kurzen Worten die *Siemenssche zwillingspathologische Vererbungsregel*, an deren Grundgedanken sich im weiten Verlauf des Ausbaus der Zwillingsmethode nichts entscheidend geändert hat. Nach wie vor sieht man in ihr das „empfindlichste Reagens für die Diagnose von Erbe und Umwelt unter den Ursachen einer Krankheit. Sie ist untrüglich und zuverlässig wie z. B. Lackmuspapier bei der Feststellung der Harnreaktion“ (*Von Verschuer 1949*).

Einwände gegen die Methode.

a) Sind EZ erbgleich?

Die erste Frage jeder Zwillingskritik ist wohl die nach der Erbgleichheit der eineiigen Zwillinge, denn das ist die Voraussetzung der Methode überhaupt. Tatsächlich sind hier auch die ersten Einwände erfolgt. Sie konnten aber schon von *Siemens* in seiner „Zwillingspathologie“ entkräftet werden, wo darauf hingewiesen wird, daß kein Argument entscheidend gegen die Genotypengleichheit eines EZ-Paars spreche. Für Genotypengleichheit spricht hingegen die große, oft verwechselbare Ähnlichkeit eineiiger Zwillinge in polymer bedingten Eigenschaften und die Übereinstimmung zwischen homologen und Rechts-Links-Verschiedenheiten bei bilateralsymmetrischen Merkmalen. Ich brauche wohl nicht näher darauf einzugehen. Besonders *von Verschuer* hat sich um die Klärung aller hieher gehörigen Fragen verdient gemacht. Es gibt nur zwei Möglichkeiten, die zu Erbunterschieden bei eineiigen Zwillingen führen können.

Das sind zufällige, während oder nach der Zwillingsbildung auftretende Chromosomen- und Genmutationen. Die ersten kommen durch Störungen der Kernteilung zustande, die letztern durch spontan erfolgende Veränderungen im Genbestand des einen Zwillingspartners. Nach Analogieschlüssen aus der Experimentalgenetik dürften sie aber so selten sein, daß praktisch nicht mit ihnen gerechnet werden muß. Die Möglichkeit ihres Auftretens widerlegt jedenfalls in keiner Weise die wohlgegründete Annahme einer Erbgleichheit der EZ.

Nicht zu verwechseln mit mutationsbedingten Diskordanzen sind die von Dahlberg (1930) hervorgehobenen und vielfach mißverstandenen „genotypischen Asymmetrien“ bilateraler Merkmale. Sie haben mit einer Erbungleichheit der EZ nicht das mindeste zu tun, obwohl sie zu Merkmalsdifferenzen führen können, die nicht auf Umwelteinflüssen im gewöhnlichen Sinne beruhen. Die Ursache scheint in zufälligen Variationen der frühesten embryonalen Entwicklungsbedingungen bzw. der engsten zytoplasmatischen Umwelt von Genen zu liegen, die Lenz (1936) als „entwicklungslabile“ Anlagen den nur „umweltlabilen“ Anlagen zur Seite stellen möchte. Es würde sich hier also um eine Eigenschaft des Gens selbst handeln, sofern man den Nachdruck mehr auf dessen autonome Reaktionsweise als auf die entwicklungsbedingte Situation, mit der sich das Gen auseinanderzusetzen hat, legt. Von Verschuer (1949) spricht deshalb von autonomer Variabilität.

b) *Lassen sich EZ mit genügender Sicherheit als solche erkennen?*

Dies ist die häufigst aufgeworfene Frage der bisherigen Zwillingskritik, da nach Meinung vieler ein Zirkelschluß des Siemenschen Ähnlichkeitsverfahrens vorliegt. Wie eingangs erwähnt, gelang die Unterscheidung der EZ von den gleichgeschlechtigen ZZ erst, als man die auf ihre Eiigkeit zu prüfenden Zwillingspaare auf die Konkordanz *mehrerer* erblich bedingter Merkmale zu untersuchen begann¹. Mit der Bestimmung des Ähnlichkeitsgrades eines Merkmals allein kam man nämlich nicht zu befriedigenden Resultaten (Poll 1914), weshalb es einen unbestreitbaren Fortschritt bedeutete, als Siemens darauf aufmerksam machte, daß eine Übereinstimmung

¹ Der meist unbekannte Eihautbefund läßt hier insofern im Stich, als man wohl von Monochorie auf Eineiigkeit, nicht jedoch von Dichorie auf Zweieiigkeit schließen kann, da je nach dem Zeitpunkt der Trennung der Zwillingshälften mono- oder dichorische EZ entstehen können. Dichorische gleichgeschlechtige Zwillinge sind demnach eineiig *oder* zweieiig.

in mehreren erbbedingten Eigenschaften nur bei genotypisch identischen Individuen (EZ) zu erwarten ist. Umgekehrt wird die Wahrscheinlichkeit, daß genotypisch verschiedene Individuen (ZZ) in mehreren Erbmerkmalen übereinstimmen, mit jedem weiteren herangezogenen Merkmal geringer.

Der Zirkelschluß des Verfahrens liegt nun darin, daß die Beweisgründe für die Eineiigkeit, nämlich die Merkmalsähnlichkeiten, selber eine Folge des zu beweisenden Urteils über die Eineiigkeit sind (Gottschick 1937a). Anders ausgedrückt: durch die Feststellung, daß ein Zwillingspaar in mehreren als erblich betrachteten Merkmalen übereinstimmt, soll die Eineiigkeit dieses Paars bewiesen werden; als eineiiges Paar muß es jedoch in allen Erbmerkmalen übereinstimmen, folglich auch in den zur Ähnlichkeitsdiagnose herangezogenen. Hier liegt tatsächlich eine *Petitio principii* vor, obwohl dies Siemens 1937 nicht wahrhaben will.

Ebenso ist die Forderung, daß die Diagnosemerkmale durch modifikatorische Einflüsse wenig veränderlich sein müssen, insofern eine Voraussetzung, als man die Modifikationsbreite einer Eigenschaft doch erst anhand der Zwillingsbefunde feststellen möchte. Nur umweltstabile Merkmale können wir in größerer Konkordanz bei EZ, in größerer Diskordanz bei ZZ anzutreffen hoffen. Bei umweltlabilen Merkmalen dagegen ist zu erwarten, daß auch einmal EZ ähnlich hohe Diskordanzgrade aufweisen, wie sie bei ZZ häufig sind. Als Resultat hätten wir dann eine nicht näher zu bestimmende Anzahl „unähnlicher“ EZ, die vermutlich als ZZ diagnostiziert würden.

Was für die umweltbedingte Variabilität der Diagnosemerkmale gilt, gilt auch für ihre Mixovariabilität. Streng genommen müßte eigentlich erst der Grad der Genotypenverschiedenheit einer Bevölkerung, der die Zwillinge entnommen sind, bekannt sein, wenn die Merkmalsunterschiede richtig bewertet werden sollen. Zum mindesten verdienen die genotypischen Verhältnisse der Familienangehörigen der Zwillinge volle Berücksichtigung¹. Bei der heute üblichen Ähnlichkeitsbestimmung wird aber eine solche Sicherung des Verfahrens praktisch nirgends angewandt.

¹ Einen Fall von großer relativer Homozygotie eines Elternpaares hat Gates 1929 beschrieben. Die Eiigkeitsbestimmung der gleichgeschlechtigen Drillinge dieser Familie bot entsprechende Diagnoseschwierigkeiten. In dem Zusammenhang sei auch das von Waardenburg 1926 publizierte Brüderpaar erwähnt, welches in den Papillarmustern aller zehn homologen Finger übereinstimmte.

So berechtigt diese Einwände sind, so gibt es nun doch einen Weg, der gestattet, mit der Ähnlichkeitsdiagnose zu genügend sichern Ergebnissen zu gelangen. Und zwar 1. durch Kontrollprüfungen an monochorischen, also sicher eineiigen Zwillingen, woraus sich ein zuverlässiger Maßstab für den Grad der zu erwartenden Übereinstimmung der Diagnosemerkmale bei EZ gewinnen ließe. Und 2. durch vermehrte Heranziehung homolog-konkordanter und spiegelbild-asymmetrischer Merkmalsähnlichkeiten, die sich infolge der besondern epigenetischen Verhältnisse der eineiigen Zwillingschwangerschaft unverhältnismäßig häufiger bei EZ als bei ZZ nachweisen lassen müßten. Schließlich kann man 3. von sicher zweieiigen Zwillingen – ungleichgeschlechtigen Paaren oder solchen mit verschiedener Blutgruppe – ausgehen und deren Variationsbreite für die einzelnen Merkmale bestimmen. Nur wenn die Ähnlichkeitsdiagnose einmal in der Weise unterbaut ist, wird es möglich sein, die ein- und zweieiigen Zwillinge einwandfrei voneinander zu trennen. Untersuchungen dieser Art liegen allerdings noch kaum vor. Bis jetzt haben einzig Stocks (1930) und Essel-Möller (1938, 1941) versucht, einen zahlenmäßigen Ausdruck für die Wahrscheinlichkeit einer bestimmten Eiigkeitsdiagnose zu berechnen.

Weist somit das Siemenssche Ähnlichkeitsverfahren gewisse Mängel auf, die kaum bestritten werden können, so darf man ihnen doch keine zu große methodologische Bedeutung beimesse. Die eigentlichen Fehler der Zwillingsmethode liegen anderswo. Es sind deshalb die ersten beiden der aufgeworfenen Fragen als im positiven Sinne entschieden anzusehen: EZ sind erbgleich und lassen sich – so nehmen wir für einmal an – mit genügender Sicherheit von ZZ unterscheiden.

c) Sind Merkmalsdifferenzen zwischen EZ und ZZ überhaupt vergleichbar?

Zu den wichtigsten genetischen Problemen, welche die Zwillingsforschung zu lösen versucht, gehört der Nachweis der Erblichkeit oder des Erblichkeitsgrades einer Eigenschaft, zu welchem Zwecke man das jeweils vorliegende Konkordanz-Diskordanzverhältnis bei EZ und ZZ miteinander vergleicht. Unter Konkordanz wird Merkmalsübereinstimmung, unter Diskordanz Merkmalsverschiedenheit der Zwillingspartner verstanden. Je nach Bedarf können 2–5 und mehr Konkordanzstufen oder Ähnlichkeitsgrade unterschieden werden. Als Resultat aus vielen hunderten derartiger Untersuchungen an Zwillingsserien ergibt sich immer wieder die in ihrer Monotonie

nachgerade langweilige Feststellung, daß die durchschnittlichen Merkmalsdifferenzen bei den EZ in der überwiegenden Mehrzahl der Fälle kleiner sind als bei den ZZ, worauf regelmäßig der Schluß auf mehr oder weniger große Erblichkeit der betreffenden Eigenschaften gezogen wird. (Vgl. auch Abschnitt d.)

Meines Wissens ist noch nie mit dem nötigen Nachdruck darauf hingewiesen worden, daß Konkordanz und Diskordanz bei EZ nicht dasselbe bedeuten wie bei ZZ. Man wird hier sofort einwenden: das sollen sie auch nicht. Die Merkmalsunterschiede der eineiigen Zwillingspaare sind allein durch Umweltfaktoren bedingt, diejenigen der zweieiigen durch Umwelt- und Erbfaktoren. Also können sie gar nicht dasselbe bedeuten. Das ist richtig. Doch übersieht man dabei, daß nicht nur danach gefragt werden kann, wie die Differenzen bei den Partnern der beiden Zwillingssgruppen zustandekommen, sondern auch, ob sie überhaupt miteinander vergleichbar sind, d. h. mit demselben Maßstab gemessen werden. Angenommen, der Ähnlichkeitsgrad einer Merkmalsdifferenz würde bei den EZ in mehrerer Hinsicht anders bestimmt als bei den ZZ, so käme dem Verhältnis der konkordanten zu den diskordanten Paaren bei den eineiigen und den zweieiigen Zwillingen offenbar eine verschiedene Bedeutung zu. Im folgenden sollen einige Punkte herausgegriffen werden, die geeignet erscheinen, den Beweis dafür zu erbringen, daß dies tatsächlich der Fall ist.

1. Es wird viel zu wenig beachtet, daß die Konkordanzerwartung bei Zwillingen nicht nur von der Art und Stärke der Umwelteinflüsse abhängt, sondern auch von der Häufigkeit, mit der das untersuchte Merkmal in der Bevölkerung auftritt. Was die Intensität der Umweltwirkung anbelangt, so wird sie, falls ihr die Paarlinge in verschiedener Weise ausgesetzt sind, größere Merkmalsunterschiede bei den ähnlichen Zwillingen hervorrufen als bei den unähnlichen, da sich bei den letztern die gleich- und ungleichgerichteten Umwelteinflüsse im Durchschnitt mehr oder weniger aufheben. *Dieselbe* peristatische Beeinflussung einer Merkmalsausbildung bewirkt also einen *verschiedenen* Diskordanzgrad in den beiden Zwillingssgruppen.

Ebenso verschiebt die Frequenz der Umweltfaktoren das Konkordanz-Diskordanzverhältnis, denn mit zunehmender Häufigkeit eines Leidens nimmt auch die Wahrscheinlichkeit des übereinstimmenden Befallenseins der Zwillingsspartner zu. Das gilt zwar für EZ und ZZ in gleicher Weise. Doch ist der Diskordanzgrad der zweieiigen Zwillinge auch noch vom Erbgang der in Frage stehenden Merkmals-

anlage abhängig, was bei den eineiigen nicht der Fall ist. Ein selten auftretendes, rezessiv vererbtes Merkmal z. B. würde einen viel höhern Diskordanzgrad aufweisen als ein selten auftretendes, dominant vererbtes. (Näheres darüber bei *Dahlberg* 1942.) Welche Art von Vererbung jeweils vorliegt, ist aber nicht von vornherein bekannt, da ja die Erblichkeit bzw. Nichterblichkeit eines Merkmals erst mit Hilfe der Zwillingsbefunde festgestellt werden soll. *Lenz* [1936] bemerkt deshalb mit Recht, daß man aus dem bloßen Diskordanzverhältnis bei EZ und ZZ den Grad der Modifizierbarkeit einer Eigenschaft nicht genauer bestimmen kann, weil es sich gewissermaßen um eine Gleichung mit mehreren Unbekannten handelt.

2. Der Konkordanzvergleich zwischen ein- und zweieiigen Zwillingen erfährt aber auch eine Beeinträchtigung durch das gegenseitige Verhältnis der allelen Gene einer Erbanlage. Nehmen wir an, ein Merkmal sei monomer bedingt und manifestiere sich mit vollständiger Penetranz. Dann entgehen bei rezessivem Erbgang sämtliche in bezug auf das Merkmal heterozygoten EZ-Partner (alle negativ-konkordanten Paare) der Beobachtung und damit dem Ähnlichkeitsvergleich, während ein Teil der heterozygoten ZZ-Partner – jene aus diskordanten Paaren – erfaßt werden, da ja der Beobachter durch das Vorhandensein des homozygoten Partners auf das Merkmal aufmerksam wird. Der durch die negativ-konkordanten Paare bedingte „Konkordanzverlust“ ist also bei den EZ größer als bei den ZZ, d. h. die Merkmalsähnlichkeiten zwischen den Paarlingen kommen in den beiden Zwillingsgruppen nicht im gleichen Maße zum Ausdruck.

Von wesentlich größerer Bedeutung für das Konkordanz-Diskordanzverhältnis scheint jedoch die Abhängigkeit der *Merkmalausprägung* vom Zustand der Heterozygotie bzw. Homozygotie der ihr zugrunde liegenden Erbanlage zu sein. Die dadurch bedingten „interpaarigen genotypischen Unterschiede“, wie *Rife* (1950) sie nennt, würden sich demnach bei homozygoten und heterozygoten Zwillingspaaren in verschiedener Weise geltend machen: „The possibility that interpair genotypic differences may also account for variations in the degree of intrapair similarity is frequently overlooked. That is to say, the extent to which a trait may be conditioned by environment is dependent on genotype. Twins heterozygous for handedness, for example, are more affected by prenatal circumstances than are those who are homozygous... It may be misleading, therefore, to estimate the role of heredity solely on the relative proportions of concordance in the two types of twins“.

3. Eine weitere Fehlerquelle ergibt sich aus der Fassung des Konkordanzbegriffes selbst. Da eineige Zwillinge denselben Genotypus besitzen, darf vorausgesetzt werden, daß sie eine geringere Merkmalsvariabilität aufweisen als die genotypisch verschiedenen zweieiigen. Es müssen deshalb die Unterschiede der ZZ im Vergleich zu denjenigen der EZ um so größer erscheinen, je höhere Anforderungen man an den Grad der Merkmalsübereinstimmung stellt. Das zeigt sich sehr schön bei fluktuierend variablen Eigenschaften, wo die quantitativ bestimmbaren Konkordanzstufen fast beliebig vermehrt (verfeinert) werden können, was regelmäßig zur Folge hat, daß die Häufigkeit der diskordanten Paare verhältnismäßig stärker bei den zweieiigen als bei den eineiigen Zwillingen zunimmt. Werden umgekehrt die Anforderungen an den Ähnlichkeitsgrad einer Merkmalsdifferenz gesenkt, so fallen nicht nur die meisten EZ in die gleiche Konkordanzstufe, sondern auch ein größerer Teil der ähnlicheren ZZ, deren Ähnlichkeitsgrad in Wirklichkeit aber keineswegs an denjenigen der eineiigen Paare heranreicht. Der Konkordanzgrad zwischen ein- und zweieiigen Zwillingspartnern wird also mit verschiedenen Maßstäben gemessen, etwa so, wie wenn ich als Anthropologe die Körperhöhe zweier Populationen das einmal auf den Zentimeter genau, das anderemal auf den Millimeter genau bestimmen würde. Solche Maße sind offenbar nicht direkt miteinander vergleichbar. Im Hinblick auf die Auswertungsmethoden erbpsychologischer Zwillingsuntersuchungen hat besonders *Wilde* (1941) die Abhängigkeit des Merkmalsvergleichs zwischen EZ und ZZ von der Wahl des Maßstabes betont.

4. Dazu kommt die Berücksichtigung des Meßfehlers, der sich bei EZ und ZZ verschieden stark auf die Merkmalsunterschiede auswirkt, und zwar im Sinne einer Vergrößerung bei den eineiigen, einer Verkleinerung bei den zweieiigen Zwillingen. Denn je kleiner die wahre Differenz zweier Maße ist – bei den genotypisch gleichen EZ von vornherein zu erwarten –, um so stärker tritt der Meßfehler in Erscheinung. Darauf hat als erster *Lenz* (1932) hingewiesen.

Ein Beispiel möge dies erläutern. Die Partner eines EZ-Paars seien in Wirklichkeit beide 150 cm groß, der Meßfehler betrage 2 % = 3 cm. Angenommen, er wirke sich mit gleicher Wahrscheinlichkeit in vier verschiedenen Richtungen aus, so messen wir entweder 1. beide Partner zu groß, 2. beide zu klein, 3. I zu groß und II zu klein, 4. I zu klein und II zu groß. Wir erhalten dann:

	Zwillingspartner	Wirkliche Körperhöhe (cm)	Meßfehler (cm)	Körperhöhe mit Meßfehler (cm)	Differenz der Partner (cm) (%)
1.	→ I	150	+ 3	153	0 = 0
	→ II	150	+ 3	153	
2.	← I	150	- 3	147	0 = 0
	← II	150	- 3	147	
3.	→ I	150	+ 3	153	6 = 4
	→ II	150	- 3	147	
4.	← I	150	- 3	147	6 = 4
	→ II	150	+ 3	153	
Im Durchschnitt					12 = 8 : 4 = 2 %

Gleichsinnig gerichtete Meßfehler machen sich also bei einer realen Differenz von 0 % nicht bemerkbar, entgegengesetzt gerichtete dafür um den doppelten Betrag, so daß sich der Meßfehler von 2 % im Durchschnitt voll auswirkt. Die mittlere Differenz von 2 % entspricht demnach nicht dem wirklichen Unterschied, der 0 % beträgt.

Anders bei großen realen Differenzen, wie sie vor allem bei zweieiigen Zwillingen zu erwarten sind. Nehmen wir an, ein ZZ-Paar habe einen wirklichen Körperhöhenunterschied von 25 cm; der eine Partner sei 150, der andere 175 cm groß. Die wahre Differenz beträgt somit 14,3 % des größeren Maßes. Bei einem Meßfehler von wiederum 2 % ergibt sich:

	Zwillingspartner	Wirkliche Körperhöhe (cm)	Meßfehler (cm)	Körperhöhe mit Meßfehler (cm)	Differenz der Partner (cm) (%)
1.	→ I	150	+ 3	153	25,5 = 14,6
	→ II	175	+ 3,5	178,5	
2.	← I	150	- 3	147	24,5 = 14,0
	← II	175	- 3,5	171,5	
3.	→ I	150	+ 3	153	18,5 = 10,6
	→ II	175	- 3,5	171,5	
4.	← I	150	- 3	147	31,5 = 18,0
	→ II	175	+ 3,5	178,5	
Im Durchschnitt					100,0 = 57,2 : 4 = 14,3 %

Die durch den Meßfehler bedingte durchschnittliche Differenz von 14,3 % ist hier nicht größer als die reale Differenz von ebenfalls 14,3 %, da sich gleich- und ungleichgerichtete Meßfehler gegenseitig aufheben. Je größer also eine wahre Merkmalsdifferenz zwischen zwei Partnern ist, um so weniger beeinflußt sie das durch den Beobachtungsfehler beeinträchtigte Resultat und umgekehrt. Der Meßfehler wirkt sich deshalb hauptsächlich bei eineiigen Zwillingen aus, deren Merkmalsunterschiede sich mehr oder weniger dem Nullwert nähern, und läßt ihren Konkordanzgrad kleiner erscheinen, als er in Wirklichkeit ist. Man darf sogar vermuten, daß viele der bei EZ gefundenen Differenzen nicht wahre Unterschiede, sondern Auswirkungen des Meßfehlers sind.

Im gleichen Sinne wird das Resultat durch die Größe des Meßfehlers selbst beeinflußt, nicht nur durch die Größe der Merkmalsdifferenz zwischen den Paarlingen. Auch hier gilt, daß die Unterschiede der eineiigen Zwillinge um so stärker betont werden, je größer der Fehler ist, den ich beim Messen mache. Die durchschnittliche Konkordanz der EZ muß deshalb zu klein ausfallen. (Ein statistisches Verfahren, das den Einfluß des Meßfehlers ausschalten soll, gibt Dahlberg 1926.)

5. Weitere Schwierigkeiten für den Konkordanzvergleich erwachsen der Zwillingsforschung aus der Deutung der Merkmalsähnlichkeiten. „Die Konkordanz bei EZ“, schreibt von Verschuer (1949) „ist wohl fast immer erbbedingt. Sie kann aber auch peripherisch bedingt sein (gleichzeitiges Auftreten derselben Infektionskrankheit)“. Ferner seien auch plasmatisch bedingte Unterschiede denkbar, z. B. bei der mongoloiden Idiotie. Nicht weniger als drei Möglichkeiten der Deutung werden uns also geboten. Der Vergleich mit dem Merkmalsverhalten der ZZ soll zwar die Entscheidung erleichtern, welche Art Konkordanz jeweils vorliegt, wenigstens was die Wahl zwischen erbbedingter und umweltbedingter Konkordanz anbetrifft. Nach welchen Kriterien man aber eine plasmatisch bedingte Merkmalsübereinstimmung beurteilen soll, wird nicht gesagt. Es befriedigt wenig, wenn versichert wird, daß es sich nur um eine selten vorkommende Ausnahme handelt.

Gerade das Beispiel der mongoloiden Idiotie zeigt schlagend, wie vieldeutig der Konkordanzbegriff im Grunde genommen ist. Man sollte denken, daß der Mongolismus nach der Faustregel der Zwillingsmethode eine „rein erbliche“ Mißbildung sei, denn alle eineiigen Zwillinge sind hier angeblich konkordant, die überwiegende Mehrzahl

der zweieiigen diskordant. Aber Luxenburger (1930), auf den die Ansicht von der plasmatisch bedingten Konkordanz der mongoloiden EZ zurückgeht, glaubt in dem Fall eine Ausnahme von der Regel machen zu müssen. Die vollkommene Merkmalsübereinstimmung erbgleicher Zwillinge sage nämlich noch nichts darüber aus, ob eine erbliche Eigenschaft vorliege. Sie beweise lediglich, daß bis zum Zeitpunkt der Trennung der Zygote die Voraussetzungen zum Auftreten des Merkmals bereits gegeben waren. Ob es sich dabei um eine Plasmaschädigung handle, könne erst mit Hilfe der Familienforschung entschieden werden. Damit wird nicht nur die Selbständigkeit der Zwillingsforschung in Frage gestellt, sondern auch ein gefährlicher Präzedenzfall geschaffen, der einer Desavouierung der Methode gleichkommt. Denn dieselbe Argumentation läßt sich natürlich auf alle angeborenen Mißbildungen anwenden, von denen jedoch mehrere als erbbedingt gelten, obwohl die betreffenden Zwillingsbefunde ähnlich lautende Diskordanzquotienten aufweisen wie bei der mongoloiden Idiotie.

Wenn von Verschuer schließlich die Diskordanz bei EZ im allgemeinen auf peristatische Einflüsse zurückführt, so ist die Frage zum mindesten erlaubt, warum er nicht auch die Konkordanz darauf zurückführt, falls es sich um gleichsinnig gerichtete Umweltfaktoren handelt. Er läßt zwar peristatisch bedingte Konkordanz zu, wie oben erwähnt, hält sie aber für viel seltener als die „wohl fast immer“ erbbedingte Konkordanz. Welche Gründe führen ihn zu dieser Annahme? Es gibt wohl keine sicheren Anhaltspunkte, die ein Abwägen der relativen Häufigkeit der gleichsinnig und entgegengesetzt gerichteten Umwelteinflüsse gestattet. Rein zufällig müßten sie etwa gleich häufig sein, vermutlich überwiegen jedoch die ersten (Lenz 1932)¹. In dem Fall ergäbe sich die paradoxe Situation, daß der größere Teil der EZ-Konkordanzen auf dieselbe Weise zustande käme wie die Diskordanzen, eben durch peristatische Momente.

Das führt zur weitern Frage, wie man sich eigentlich eine erbbedingte Konkordanz bei EZ vorzustellen hat. Indem man von sämtlichen Umweltfaktoren abstrahiert und das Gen allein für die Merkmalsübereinstimmungen verantwortlich macht? Oder spielt die Peristase doch eine Rolle, ähnlich wie bei den Diskordanzen? Etwa

¹ Selbst wenn man nicht so weit gehen will wie von Uxküll (1921), der die Umwelt geradezu als eine Funktion des Organismus (d. h. doch wohl des Genotypus) bezeichnet, wird man zugeben müssen, daß eineiige Zwillinge im allgemeinen in der denkbar ähnlichsten Umweltgemeinschaft aufwachsen.

in dem Sinne, daß *jedes* Merkmal – und folglich auch jeder Merkmalsunterschied zwischen den Paarlingen, sei er nun groß oder klein, diskordant oder konkordant – das Reaktionsergebnis von Gen und Umwelt ist? Die Beantwortung dieser Frage hängt offenbar davon ab, was man unter erblich und nichterblich versteht. Ich komme noch darauf zurück.

6. Eine andere Fehlermöglichkeit liegt darin, daß das Konkordanz-Diskordanzverhältnis von der Größe der Manifestationsschwankung bzw. der Modifizierbarkeit eines Merkmals beeinflußt wird, von Umständen also, die doch erst mit Hilfe der Zwillingsforschung bestimmt werden sollten (Zirkelschluß). Man kann nicht beides zugleich tun: den Grad der Erblichkeit eines Merkmals bestimmen wollen und gleichzeitig die Modifizierbarkeit eben dieses Merkmals. Das führt nur zu einer Konfusion des Erbe-Umweltbegriffs, wovon später noch die Rede sein wird. Darüber hinaus sollte man die Möglichkeit „genotypischer Asymmetrien“ bilateraler Merkmale nicht vergessen, die bei EZ – und nur bei diesen – Manifestationsschwankungen vortäuschen können, welche mit Umweltwirkungen im gewöhnlichen Sinne nichts zu tun haben. (Vgl. Abschnitt a.) Da diese autonomen Manifestationsstörungen keineswegs selten sein sollen (Lenz 1936), bilden sie eine weitere, ernst zu nehmende Fehlerquelle der Konkordanzbestimmung. Wenn von Verschuer glaubt, die autonome von der peristatisch bedingten Variabilität wenigstens schätzungsweise trennen zu können, indem er die durchschnittlichen Differenzen umweltähnlicher und umweltverschiedener EZ miteinander vergleicht, so widerspricht er seinen eigenen Angaben. Betont er doch ausdrücklich, daß sich „selbst bei gleicher Umwelt“ verschiedene Möglichkeiten unvollständiger autonomer Manifestierung verwirklichen können und damit zu Unterschieden bei genotypisch identischen Zwillingen führen. Der andere Vorschlag, die ungefähre Größe der *random variation* zu bestimmen – Vergleich der durchschnittlichen Paarverschiedenheit der EZ mit der durchschnittlichen Verschiedenheit der Körperhälften eines Paarlings –, erweckt ähnliche Bedenken, worauf übrigens von Verschuer selbst hinweist. Auch Dahlberg (1950) hält eine einwandfreie Trennung von autonomer und peristatischer Variabilität nicht für möglich.

7. Schließlich wäre noch der Geschlechtsunterschied zu berücksichtigen, der sich bei jenen Merkmalen geltend macht, die das eine Geschlecht ausschließlich oder vorwiegend befallen. Bei einem Leiden

mit geschlechtsgebundenem Erbgang (Hämophilie) oder völliger Geschlechtsbegrenztheit (Hypospadie) werden Pärchenzwillinge immer diskordant sein, weshalb man den Vergleich nur innerhalb der Zwillingspaare mit gleichem Geschlecht durchführen darf. Auch bei teilweise geschlechtsbegrenzten Merkmalen – und hieher gehören eine ganz beträchtliche Anzahl normaler und pathologischer Eigenschaften – ist unter den gleichgeschlechtigen ZZ von vornherein eine größere Konkordanz zu erwarten als unter den PZ. Solche Merkmalsvergleiche müssen deshalb immer mit gewissen „Konkordanzverlusten“ bei den Zweieiigen rechnen, sofern man gleich- und ungleichgeschlechtige ZZ nicht getrennt behandelt.

8. Genau wie das Geschlecht kann auch der jeweilige Entwicklungszustand eines Merkmals, also dessen Altersabhängigkeit, einen Einfluß auf den Konkordanzvergleich ausüben. So wird z. B. bei einem Leiden mit großer Manifestationsbreite bzw. langer Gefährdungsperiode der Diskordanzgrad der EZ um so höher ausfallen, je kürzer die Zeitspanne ist, innerhalb welcher die merkmalsfreien Zwillingspartner beobachtet werden. Anderseits muß die Anzahl der diskordanten ZZ-Paare insofern zu niedrig erscheinen, als ein Teil derselben überhaupt nicht erfaßt werden kann, wenn die Beobachtungszeit zu kurz angesetzt wird. Nicht überall steht ein relativ so zuverlässiges Kriterium wie beim Diabetes mellitus zur Verfügung (pathologische Blutzuckerkurve des noch merkmalsfreien Partners nach Dextrosebelastung), mit dessen Hilfe sich die manifestationsverhinderten konkordanten EZ-Paare als solche erkennen lassen.

Auf Grund dieser und anderer, hier nicht weiter zu erörternder Überlegungen kommt man zum Resultat, daß Konkordanz und Diskordanz bei EZ und ZZ nicht dasselbe bedeuten, weil die Merkmalsunterschiede in den beiden Zwillingsgruppen mit verschiedenen Maßstäben gemessen werden. Es werden gewissermaßen unvergleichbare Größen miteinander verglichen. Man hat auch den Eindruck, daß zu viele unbekannte Faktoren vorliegen, von denen der Ähnlichkeitsgrad einer Merkmalsdifferenz abhängt, weshalb zu viele Voraussetzungen gemacht werden müssen. Eine richtige Beurteilung der Befunde wird dadurch erschwert, wenn nicht verunmöglich. Das ist nun insofern fatal, als sich die Zwillingsforschung ausschließlich der Konkordanz-Diskordanzverhältnisse für ihre Schlüsse bedient. Ihre Ergebnisse sind deshalb von zweifelhaftem Wert, zumal es noch andere Gesichtspunkte gibt, von denen aus sich die Grundlagen der

Methode kritisch beleuchten lassen. Methodologisch fallen sie sogar noch schwerer ins Gewicht als die eben vorgebrachten Einwände. Ich begnügen mich im folgenden mit einigen kurzen Hinweisen.

d) Läßt sich der relative Anteil von Erbe und Umwelt an der Entstehung eines Merkmals bestimmen?

Einleitend wurde die sogenannte „zwillingspathologische Vererbungsregel“ von Siemens zitiert, nach der ein erblich bedingtes Merkmal bei EZ häufiger gemeinsam angetroffen wird bzw. einen höheren durchschnittlichen Ähnlichkeitsgrad besitzt als bei ZZ. Umgekehrt ist bei diskordantem Merkmalsverhalten der EZ besonders dann auf Nichterblichkeit zu schließen, wenn die betreffende Eigenschaft auch bei ZZ einen hohen Diskordanzgrad aufweist. Am Ähnlichkeitsquotienten der beiden Zwillingssgruppen mißt man also die Erblichkeit oder den Erblichkeitsgrad der daraufhin untersuchten Merkmale und fragt, *wie weit* die und die Eigenschaft durch Erbinflüsse und Umwelteinflüsse bestimmt wird.

Dabei argumentiert man folgendermaßen: Merkmalsunterschiede eineriiger Zwillinge sind durch die Umwelt bedingt, Merkmalsunterschiede zweieiiger Zwillinge durch die Umwelt *und* durch die Erbmasse. Setzt man voraus, daß der umweltbedingte Unterschied bei EZ und ZZ gleich groß ist – Zwillingsspartner wachsen im allgemeinen in „derselben“ Umwelt auf –, so erhält man die erbbedingte Merkmalsdifferenz zweieiiger Zwillinge durch einfache Subtraktion des Merkmalsunterschiedes der EZ von demjenigen der ZZ. Als Formel dargestellt: U bedeutet einen Merkmalsunterschied überhaupt, u den umweltbedingten und e den erbbedingten Anteil daran. Dann ist

$$U_{EZ} = \text{umweltbedingt}$$

$$U_{ZZ} = \text{umweltbedingt } (U_{ZZu}) + \text{erbbedingt } (U_{ZZe})$$

Voraussetzung: $U_{EZ} = U_{ZZu}$

also $U_{ZZ} = U_{EZ} + U_{ZZe}$

daraus $\underline{U_{ZZe} = U_{ZZ} - U_{EZ}}$

U_{ZZe} wäre somit der erbbedingte Anteil bzw. der Grad der Erbbedingtheit des daraufhin untersuchten Merkmals.

Die Überlegung, welche dieser scheinbar einwandfreien Schlußfolgerung zugrunde liegt, beruht jedoch auf einer unrichtigen Anwendung der Terminologie, die im Jahre 1909 von Johannsen zur

Bestimmung des Erbe-Umweltbegriffs in die Genetik eingeführt wurde und heute noch ihre Gültigkeit besitzt. *Johannsen* operiert mit den drei Grundbegriffen Genotypus (Erbbild), Phänotypus (Erscheinungsbild) und Lebenslage (Umwelt). Mit gegebenem Genotypus ist die Gesamtheit der Entwicklungsmöglichkeiten, die Reaktionsweise der Zygote, bestimmt. Was von diesen Möglichkeiten verwirklicht werden soll, hängt von der jeweiligen Lebenslage ab. Das Resultat der Auseinandersetzung des Genotypus mit seiner Umwelt ist die Gesamterscheinung des betreffenden Organismus, der Phänotypus. *Johannsen* gibt dafür das Schema:

$$\begin{array}{c} \text{Genotypus mit Lebenslage} \\ \hline \text{Phänotypus} \end{array} \quad (1)$$

$$\text{oder auch: Phänotypus} \sim \text{Genotypus} + \text{Lebenslage} \quad (2)$$

Der Phänotypus ist somit von zwei verschiedenartigen, in engster Beziehung zueinander stehenden Variablen abhängig, der Erbanlage einerseits und der Umwelt andererseits. Aber es ist nicht so, daß man nach „Formel“ (2) den genotypisch bedingten Anteil eines Merkmals dadurch erhält, daß man den umweltbedingten Merkmalsanteil vom Phänotypus *subtrahiert*, wie *Siemens* anzunehmen scheint. Offenbar verwechselte er das Kongruenzzeichen mit einem Gleichheitszeichen, denn nur aus der Gleichung *Phänotypus* = *Genotypus* + *Lebenslage* ließe sich, so paradox es klingt, der Genotypus durch Subtraktion erhalten: *Genotypus* = *Phänotypus* — *Lebenslage*.

Diese merkwürdige Formulierung findet man tatsächlich in den Arbeiten der verschiedensten Autoren, z. B. noch in einer neueren Zwillingsstudie von *Schinz* (1945). Sie basiert auf der irrtümlichen Vorstellung, daß sich Erb- und Umwelteinflüsse bei der Entstehung eines Merkmals summieren, während sie sich in Wirklichkeit kombinieren, wie *Lenz* (1935) richtig bemerkt hat¹. Nur so ist es zu verstehen, daß *Siemens* in der Terminologie *Johannsens* einen Begriff für das „Nichterbliche“ eines Merkmals vermissen konnte. Er nennt ihn Paratypus oder Nebenbild und faßt ihn als den nichterblich bedingten Teil des Phänotypus auf. Im Bestreben nämlich, den lamarckistischen Gedanken einer „Vererbung erworberer Eigenschaften“ auch terminologisch zu bekämpfen, will *Siemens* die Begriffe

¹ Zu ähnlichen Überlegungen gelangte *Hogben* (1933). Vergleiche auch seine mathematische Darstellung des Problems aus dem Jahre 1951.

des Erblichen und Nichterblichen auf das schärfste voneinander getrennt wissen. Er hält es darum für verfehlt, wenn man, wie allgemein üblich, die Mutationen als genotypische Erscheinungen den Modifikationen als phänotypischen Erscheinungen gegenüberstellt. Man müsse vielmehr die Modifikationen als *rein phänotypische*, d.h. also paratypische Variabilitätserscheinungen von der Gesamtheit des Phänotypus abtrennen, weshalb er sie Paravariationen nennt (1917). Die Paravariationen haben sich in der Folge als Synonyma der Modifikationen im deutschen Schrifttum eingebürgert und weiter nicht geschadet. Der Paratypus dagegen hat bis heute eine unheilvolle Rolle in der Humangenetik gespielt und entscheidend zur Begriffsverwirrung der Zwillingsforschung beigetragen. Ohne ihn wäre die Entwicklung einer „zwillingspathologischen Vererbungsregel“ überhaupt nicht möglich gewesen.

Bereits *Johannsen* (1926) hat den Nebenbegriff des Paratypus, der den Gedankengängen der vormendelistischen Zeit zu entstammen scheint, als unnötig komplizierend zurückgewiesen. „Für das Nichterbliche einen besondern Begriff – Paratypus – zu bilden“, schreibt er, „dürfte aber kaum notwendig sein. Denn alles, was zum Phänotypus gehört, d. h. alle Eigenschaften, die am Organismus beobachtet werden können, sind *als solche* nicht erblich! Doch sind sie stets Ausdrücke direkter oder indirekter Reaktionen des Genotypus mit den Faktoren der Lebenslage. *Siemens'* Wort Paratypus scheint ausdrücken zu sollen, daß Charaktere existieren, die nichts mit dem Genotypus des betreffenden Organismus zu tun hätten, insofern also ‚rein phänotypisch‘ sein sollten. Solche Eigenschaften eines Organismus kommen aber überhaupt nicht vor: nichts ist ‚rein phänotypisch‘, weil Phänotypen notwendigerweise Reaktionen des Genotypus sind“.

Im Begriffstrio *Johannsens* ist also einzig der Phänotypus als Merkmal faßbar. Die beiden andern Begriffe, Genotypus und Lebenslage, bezeichnen zwar ebenfalls reale Gegebenheiten, können aber nur aus ihren Wirkungsweisen erschlossen werden. Sie treten uns nicht als Substanzen, sondern als biologische Kräfte entgegen. Und da sie in engster, wechselseitiger Bedingtheit aufeinander einwirken, ist es unmöglich, ihren beiderseitigen Anteil am Wirkungseffekt (dem Phänotypus) quantitativ abzugrenzen. Von der Umwelt aus gesehen kann der Genotypus nur so weit zur Merkmalsmanifestation führen, als es die vorhandenen Umweltfaktoren zulassen. Vom Genotypus aus gesehen vermag die Umwelt nur insofern in Aktion zu

treten, als es die genotypische Reaktionsweise gestattet. Beide Faktoren bestimmen sich gegenseitig. Es ist deshalb völlig sinnlos, ein Stück „Nur-Umweltbedingtheit“ vom Merkmal abziehen zu wollen – den Phänotypus vom Paratypus zu entkleiden, wie sich *Korkhaus* (1939) ausdrückt –, um zu einer meßbaren Vorstellung von der ihm zugrundeliegenden Erbanlage zu gelangen. Nichts anderes tun aber *Siemens* und mit ihm die Zwillingsforschung, wenn sie den Anteil der Erb- und Umwelteinflüsse am Zustandekommen einer Eigenschaft für sich gesondert berechnen möchten. Hier gibt es nichts zu trennen. Es gibt kein Mehr-oder-weniger an Erblichkeit, keine Grade der Erbbedingtheit eines Merkmals. Das sind sehr mißverständliche Ausdrücke. Jedes Merkmal ist das Ergebnis des Zusammenwirkens der Gene mit den Umweltfaktoren, gleichgültig, in wie weiten Grenzen es variiert. Will man seine Variabilität bezeichnen, so begnüge man sich mit Ausdrücken wie Modifizierbarkeit oder Manifestationschwankung. Diese sind es in Wirklichkeit, die von der Zwillingsforschung annähernd bestimmt werden können, und nicht die relative Erbbedingtheit einer Eigenschaft. Hier liegt die einzige fruchtbare Anwendungsmöglichkeit der Zwillingsmethode.

Allerdings kommen wir auch hier nicht um eine bedeutsame Einschränkung herum. Die an eineiigen Zwillingen bestimmbare Modifizierbarkeit, die als Merkmalsunterschied zwischen den Paarlingen zutage tritt, ist nämlich von der Genotypenverschiedenheit und der Umweltverschiedenheit innerhalb der Bevölkerung abhängig, der die Zwillinge entnommen sind. Ein eineiiges Zwillingspaar ist zwar das Analogon einer reinen Linie (besser: eines Biotypus), aber hundert eineiige Paare sind ebenso viele verschiedene reine Linien, also ein Liniengemisch. Das wird in der Regel übersehen. Jede einzelne dieser reinen Linien reagiert gemäß *ihrem besonderen Genotypus* auf die jeweils gegebenen Umweltfaktoren. Der Grad der übereinstimmenden Reaktionsweise der hundert reinen Linien hängt also vom Grad der Genotypenunterschiede (Heterogenie) zwischen ihnen ab und damit vom Grad der Heterogenie der Bevölkerung, aus der die reinen Linien stammen. Dabei ist die Voraussetzung gemacht, daß die Umweltbedingungen die gleichen sind. Sind sie verschieden, was gewöhnlich der Fall ist, so hängt der Grad der übereinstimmenden Reaktionsweise der hundert reinen Linien auch vom Grad der Variabilität der Umweltfaktoren in der betreffenden Bevölkerung ab. Die Modifizierbarkeit einer Eigenschaft, wie sie an eineiigen Zwillingen bestimmt werden kann, gilt also immer nur für die jeweils vorliegenden Erb-

und Umweltverhältnisse des Ausgangsmaterials. Davon wird nochmals die Rede sein.

Auf etwas anderm Wege kommt Lenz (1935) zum gleichen Ergebnis, ohne jedoch die letzte Konsequenz daraus zu ziehen. Vielleicht ist das der Grund, weshalb sein kritischer Einwand so wenig Beachtung fand. Wie bereits erwähnt, machte er darauf aufmerksam – was auch schon die richtige Lesung der „Formel“ Johannsens ergibt –, daß man den Anteil von Erbe und Umwelt am Zustandekommen eines Merkmals nicht exakt bestimmen könne, da sich Erbeinflüsse und Umwelteinflüsse nicht summieren, sondern binomisch kombinieren würden. Folglich lasse sich der erbbedingte Unterschied der ZZ in bezug auf ein Merkmal nicht dadurch erhalten, daß man den Unterschied der EZ von dem der ZZ subtrahiere¹. Aber die einzige richtige Schlußfolgerung aus dieser Erkenntnis, daß dann offenbar die ganze Fragestellung verfehlt sein müsse, hat auch Lenz nicht gezogen. Denn er hält es weiterhin für möglich, aus den Zwillingsbefunden wenigstens ein „Mindestmaß des Erbeinflusses“ berechnen zu können. Mit andern Worten: es soll sich der Erblichkeitsgrad eines Merkmals zwar nicht genau, aber doch ungefähr bestimmen lassen. Damit ist der logische Fehler natürlich nicht behoben.

Er wird auch nicht durch die Relativierung des Begriffs der Erblichkeit behoben, wie Lenz im Anschluß an Siemens zu glauben scheint. Der Erblichkeitsbegriff, so führt er aus, beziehe sich entweder auf die Merkmale *als solche* oder auf die Merkmalsunterschiede innerhalb einer bestimmten Bevölkerung. „Man muß sich stets klar zu werden versuchen, was man im speziellen Fall mit dem Wort „erblich“ meint. Ob einen Anteil an den tatsächlichen Unterschieden oder die genetische Grundlage des Wesens selber“. Ähnlich bekommt für Siemens (1925) die Frage nach der Erbbedingtheit „erst dann einen vernünftigen Sinn, wenn sie Unterschiede zwischen den verschiedenen Menschen ätiologisch erklären soll. Was jeder Mensch hat, ist als Artcharakter selbstverständlich erbbedingt“. Man fragt also das einmal, ob z. B. die Körperhöhe an sich erblich ist, und das anderemal, in welchem Maße die Körperhöhenunterschiede innerhalb einer Population erbbedingt sind. Unterscheiden sich im letztern

¹ Im Widerspruch dazu heißt es jedoch einige Abschnitte später: „Wenn in bezug auf eine Eigenschaft der Unterschied von EZ an genügend großem Material gleich dem von ZZ gefunden werden würde, so spräche das nach wie vor dafür, daß die Unterschiede in der betreffenden Bevölkerung rein umweltbedingt seien“. Als Formel ausgedrückt: $U_{ZZ} = U_{ZZ} - U_{EZ} = 0$.

Fall die Großen, Mittelgroßen und Kleinen durch ihren verschiedenen Genotypus oder durch die verschiedene Umwelt, in der sie leben, oder durch beide?

Was die Genotypenvariabilität anbelangt, so differieren die Merkmalsträger in einer relativ homogenen Bevölkerung offenbar weniger als in einer heterogenen, gemischtrassigen. Trotzdem ist die Körperhöhe in der einen Population genau so erblich wie in der andern. Die Zwillingsbefunde würden aber im ersten Fall eine geringere Erblichkeit der Körperhöhe anzeigen als im zweiten Fall, da gleichgroße Unterschiede bei EZ und ZZ Umweltbedingtheit, verschiedengroße Unterschiede Erbbedingtheit eines Merkmals bedeuten. Ähnlich beeinflußt die Variabilität der Umweltfaktoren innerhalb einer bestimmten Bevölkerung die Ergebnisse der Zwillingsmethode. Es können sich deshalb die Schlüsse aus dem Konkordanzvergleich „immer nur auf die tatsächlich vorkommenden Unterschiede in einer Bevölkerung von gegebener Mischung von Erbmasse und Umwelt beziehen“ (Lenz 1936). Anders ausgedrückt: die Zwillingsmethode vermag nur etwas über den Grad der Heterogenie und der Umweltverschiedenheit einer Bevölkerung auszusagen, wobei zu beachten ist, daß ihr Aussagewert mit zunehmender Reinrassigkeit und Umweltgleichheit geringer wird. Gottschick (1937b), der dieselbe Auffassung vertritt, möchte sie überhaupt nur als Methode der Rassenbiologie gelten lassen.

Ich habe den Eindruck, daß Lenz mit seiner Relativierung des Erblichkeitsbegriffes der Zwillingsforschung einen Ausweg aus dem Dilemma zeigen wollte, in das sie durch eine falsche Fragestellung geraten ist. Er möchte ihre bisherigen Ergebnisse, die nicht zuletzt durch seine Methodenkritik fragwürdig geworden sind, in einem Sinne interpretieren, der den terminologischen Anforderungen der modernen Genetik besser entspricht. Zu diesem Zwecke bietet er ihr einen neuen Erblichkeitsbegriff an. Die Zwillingsforscher haben ihn jedoch nicht akzeptiert¹. Sie haben scheinbar kein Interesse daran, die Variabilität eines Merkmals innerhalb einer gegebenen Bevölkerung daraufhin zu untersuchen, ob sie durch Unterschiede der Genotypen oder durch Unterschiede der Umweltbedingungen hervorgerufen wird. Nach wie vor bestimmen sie den Erbe-Umwelt-

¹ Lenz darf sich darüber nicht wundern. Er selbst entscheidet sich für die alte Wortbedeutung von „erblich“, wenn er schreibt: „Es liegt auf der Hand, daß der Begriff der Erblichkeit nicht mit dem der Heterogenie einer Population gleichgesetzt werden darf“ (1939).

anteil am Zustandekommen einer Eigenschaft schlechthin und glauben, damit eine Aussage über deren geringere oder größere Erblichkeit machen zu können. In Wirklichkeit lässt sich überhaupt nichts darüber aussagen, weil die Frage, wie nochmals hervorgehoben sei, falsch gestellt ist: nicht Erbe *oder* Umwelt bestimmen in verschiedenem Ausmaß ein Merkmal – so müßte man nämlich im Sinne von Siemens formulieren –, sondern Erbe *und* Umwelt in engster, untrennbarer Wechselwirkung. Jeder Versuch, den Grad der Erbbedingtheit einer Eigenschaft feststellen zu wollen, wird damit illusorisch, gleichgültig, ob er nur schätzungsweise oder auf Prozente genau bestimmt werden soll. Man darf sich den Phänotypus nicht als ein Etwas vorstellen, *an dem* Erb- und Umwelteinflüsse in verschiedener Weise angreifen. Das würde bedeuten, daß Genotypus und Phänotypus Antithesen sind, was Johannsen natürlich nicht gemeint hat.

In diesem Mißverständnis liegt der Hauptfehler der Zwillingsmethode. Er wird erst dann behoben sein, wenn man sich zu einer korrekten Handhabung des Erbe-Umweltbegriffs entschließt, wie sie in der experimentellen Genetik üblich ist. Das Mißverständnis wird aber nicht dadurch beseitigt, daß man Eigenschaften mit geringer Modifizierbarkeit als erblich bzw. stark erblich und solche mit großer Modifizierbarkeit als nichterblich oder schwach erblich bezeichnet (Siemens 1919, Lenz 1941). Das wäre nur eine weitere Konzession an den Siemensschen Paratypus, auf den letzten Endes die Verwirrung zurückgeht, welche durch die Zwillingsforschung in die klare Terminologie Johannsens gebracht wurde. Man kommt dem methodologischen Fehler auch nicht bei, wenn man den Erblichkeitsbegriff den verschiedenen genetischen Methoden anpaßt, wie Lenz (1948) es möchte. Man muß vielmehr die Konsequenzen ziehen und sich für ein und denselben Begriff entscheiden, der auf die gesamte Erbforschung anwendbar ist.

Es ließen sich noch andere Einwände anführen, mit denen man beweisen kann, daß das Erbe-Umweltproblem von der Zwillingsforschung falsch angepackt wurde. Die obigen Überlegungen sollten jedoch genügen. Nur auf eines sei noch kurz hingewiesen, auf die Behauptung nämlich, daß man die Umwelt der eineiigen Zwillinge derjenigen der zweieiigen gleichsetzen könne. Dem widerspricht schon die viel engere Zwillingsgemeinschaft der EZ, die besonders den Erbpsychologen aufgefallen ist und sie veranlaßt hat, neben dem individualistischen Ich-Erleben des einzelnen Partners auch das

sogenannte „Wir-Erlebnis“ des Paars zu berücksichtigen. Diese enge psychische Zwillingsverbundenheit hat ihre genaue Parallel im Bereich des Physischen. Wenn man davon ausgeht, daß die Umwelt eines Lebewesens weitgehend von seinem Genotypus bestimmt wird – hier kann an den Umweltbegriff von *Uxkülls* angeknüpft werden –, so versteht man, weshalb EZ-Partner eine viel ähnlichere Umwelt besitzen müssen als ZZ-Partner. Nach von *Uxküll* schafft sich jeder Organismus seine ihm allein gemäße Umwelt, die nur ein Ausschnitt aus der Umgebung des betreffenden Lebewesens ist. Identische Zwillinge verfügen deshalb immer nur über die gleichen potentiellen Möglichkeiten der Umweltvariabilität, nichtidentische Zwillinge haben dagegen verschiedene Möglichkeiten. Auch diese Betrachtungsweise läßt den Konkordanzvergleich als äußerst fragwürdige Ausgangsbasis für die Bestimmung des relativen Kräfteverhältnisses zwischen Erb- und Umwelteinflüssen erscheinen.

Ergebnis.

Niemand wird einen ernstlichen Einwand dagegen erheben können, wenn man sagt, daß die Ergebnisse einer Methode von ihren begrifflichen Voraussetzungen abhängen. Sind die Voraussetzungen falsch, so sind es auch die Ergebnisse. Im Falle der Zwillingsmethode trifft das weitgehend zu. Die Schlußfolgerungen, die wir aus unsren Darlegungen ziehen müssen, sind deshalb vorwiegend negativer Natur. Als Methode der menschlichen Erblehre ist die Zwillingsforschung in der Tat nur sehr beschränkt brauchbar. Das röhrt daher, daß sie zum Teil noch mit vormendelistischen Begriffen arbeitet, wie das Beispiel des mißverstandenen Erbe-Umweltpblems zeigt. Fast alle Resultate, welche sich auf dieses Problem beziehen, vermögen heute nur noch historisches Interesse zu erwecken. Eine Ausnahme machen einzig die Untersuchungen über die Variabilitäterscheinungen der Manifestationswahrscheinlichkeit und Modifizierbarkeit. Der Anteil von Erbe und Umwelt an der Entstehung einer Eigenschaft, das zentrale Anliegen der bisherigen Zwillingsforschung, kann jedenfalls nicht bestimmt werden. Zur Zeit Galtons mochte eine solche Fragestellung ihre Berechtigung haben. Heute ist sie überholt.

Zusammenfassung.

Es wird darauf hingewiesen, daß die Zwillingsmethode von begrifflichen Voraussetzungen ausgeht, die teilweise im Widerspruch zur Terminologie der experimentellen Genetik stehen. Das gilt vor

allem für den Versuch, das relative Kräfteverhältnis von Erbe und Umwelt am Zustandekommen eines Merkmals bestimmen zu wollen. Zudem hängt der Vergleich der Merkmalsunterschiede zwischen ein- und zweieiigen Zwillingspaaren, wie er zur Feststellung des Erblichkeitsgrades einer Eigenschaft benutzt wird, von so vielen Bedingungen ab, daß er streng genommen nicht durchführbar ist. Als Methode der menschlichen Erblehre kann die Zwillingsforschung deshalb nur in beschränktem Maße verwendet werden.

Résumé.

La méthode gémellaire part des conditions logiques qui contredisent partiellement celles de la génétique expérimentale. Cela est surtout le cas quand il s'agit de décider de l'importance relative de l'hérédité et du milieu pour la manifestation d'un caractère. Pour établir l'hérédité d'un caractère on compare les différences entre des jumeaux uniovulaires avec celles des jumeaux biovulaires. Cependant ces différences sont conditionnées par tant de circonstances que la méthode n'est pas rigoureusement applicable. Dans la génétique humaine, la méthode gémellaire n'a donc qu'un emploi limité.

Summary.

It is referred to the fact that the twin method starts from logical conditions which partly contrast with experimental genetics. This is especially the case when it is a question of deciding the relative importance of heredity and environment for the appearance of a character. Besides, the comparison of differences between monozygotic and dizygotic pairs of twins when used for establishing the heredity of a character depends on so many conditions that it cannot be carried out strictly. As a method in human genetics twin studies therefore can be used only to a limited extent.

LITERATUR

- Dahlberg, G.:* Twin births and twins from a hereditary point of view. Diss. Uppsala 1926. — *Id.:* Z. ind. Abstammungsl. 53, 133, 1930. — *Id.:* Hereditas 28, 409, 1942. — *Id.:* Acta genetica 1, 104, 1950. — *Essen-Möller, E.:* Arch. Rassenbiol. 32, 1, 1938. — *Id.:* Hereditas 27, 1, 1941. — *Galton, F.:* J. Anthr. Inst. London 5, 391, 1876 (Deutsche Übersetzung: Erbarzt 2, 132, 1935). — *Gates, R.:* J. Hered. 20, 209, 1929. — *Gottschick, J.:* Arch. Rassenbiol. 31, 185, 1937a; 31, 377, 1937b. — *Hogben, L.:* Nature and nurture. New York 1933. — *Id.:* Acta genetica 2, 101, 1951. — *Johannsen,*

W.: Elemente der exakten Erblichkeitslehre. Jena 1909. – *Id.*: Allgemeine Vererbungslehre. In: Biologie der Person (*Brugsch-Lewy*) I, 227, 1926. – *Korkhaus, G.*: Die Zwillingsforschung und ihre Bedeutung für die Erbforschung der Gebißmerkmale. In: *Hbch. Zahnhkde (Bruhn)* IV, 146, 1939. – *Lenz, F.*: Z. ind. Abstammungsl. 62, 153, 1932; *Id.*: Dt. med. Wschr. 61, 873, 1935. – *Id.*: Die Zwillingsmethode. In: *Baur-Fischer-Lenz*, Menschliche Erblehre und Rassenhygiene. 4. Aufl., München I, 641, 1936. – *Id.*: *Fortschr. Fortschr.* 15, 422, 1939. – *Id.*: Proc. 7. int. Genet. Congr. Edinburgh. Cambridge, 187, 1941. – *Id.*: Grenzgeb. Med. 1, 135, 1948. – *Id.*: Z. Vererb.-u. Konst.lehre 29, 820, 1950. – *Luxenburger, H.*: Nervenarzt 3, 385, 1930. – *Poll, H.*: Z. Ethnol. 46, 87, 1914. – *Siemens, H.*: Arch. Rassenbiol. 12, 257, 1917. – *Id.*: Dt. med. Wschr. 45, 1302, 1919. – *Id.*: Die Zwillingspathologie. Berlin 1924. – *Id.*: Sitzber. Ges. Morph. Phys. München 36, 39, 1925. – *Id.*: Arch. Rassenbiol. 31, 211, 1937. – *Rife, D.*: Hum. Biol. 22, 136, 1950. – *Schinz, H.*: Arch. Klaus-Stiftg. 20, 13, 1945. – *Stocks, P.* und *M. Karn*: Ann. Eug. 4, 49, 1930. – *Üxküll, J. v.*: Umwelt und Innenwelt der Tiere. 2. Aufl., Berlin 1921. – *Verschuer, O. v.*: S. A. S. (Bull. Com. int. Standard. anthrop.) 19, 23, 1949. – *Waardenburg, P.*: Klin. Wschr. 5, 2115, 1926. – *Wilde, K.*: Arch. Psychol. 109, 1, 1941.

From the Stomatological Clinic of the Medical University in Debrecen, Hungary
(Head: P. Adler, M. D., Assistant Professor of Orthodontics)

STUDIES ON THE ERUPTION OF PERMANENT TEETH

II. The Sequence of Eruption

By PETER ADLER and ELISABETH GÖDÉNY

In the first report on our studies it was pointed out that in regard to the time of the eruption of the individual teeth, the usual statistical significance test, using a deviation from the mean exceeding 3σ , does not seem suitable for practical clinical use. Clinical experience nevertheless seems to indicate that divergencies in the eruption of the permanent teeth might offer some criteria for determining the biological age, at least in clinical dentistry, especially if the eruption of any single tooth is not taken into consideration but the eruption of all teeth of the permanent dentition, covering a long period of juvenile life. For this reason it seems justified to seek continually for characteristics of the permanent dentition that might be used for such purposes.

Besides the mean age at the eruption of the different teeth and the number of permanent teeth erupted at specified ages, the sequence of the eruption of the permanent teeth also deserves to be studied. Attempts to such studies were made by Steggerda and Hill [1942], and by Hellman [1943]; however, in these studies, the authors being concerned mainly with differences between the eruption of the upper and the lower teeth, between girls and boys, and between different population groups and races, the sequence of eruption was determined for the upper and the lower teeth separately. In our opinion it seems self-evident that it might offer some advantages to consider the two dental arches as one unit. In the present paper studies on this theme are reported.

Material and methods. Our report is mainly based on those 8333 children from the Hungarian lowland between seven and fourteen

years of age whose tooth eruption data were reported in the first paper by Gödény [1951]. The mean (or median) age at the eruption of each tooth was determined from its presence in 50 per cent of the total number of children. The data of these children were recorded graphically and then compared with

1. data for children from other regions in Hungary than the lowland, viz.

a) the school population, born and continuously resident in one of the following seven small villages in the most northern part of the Eperjes-Tokaj mountains in the present area of Hungary: Gönc, Kéked, Telkibánya, Göncruszka, Vizsoly and Vilmány, with a total of 1083 children. This group is later simply referred to as the inhabitants of Gönc and adjacent villages ;

b) the school population, born and continuously resident in the small Transdanubian city Szekszárd, with a total of 803 children.

2. data in dental literature on the mean age at the eruption of the permanent teeth in different populations in so far as data for boys and girls are given separately.

Since the standard deviations of the mean ages at the eruption of the permanent teeth are rather considerable, it seemed advisable to us to construct graphs, taking into consideration not only the mean age at the eruption of the individual teeth but also the standard deviations of the means. In this manner consideration is paid to variations of age at the eruption of the individual teeth. Since 3σ seemed to be not quite suitable clinically, variations of $\pm\sigma$ only were taken into consideration in the graphs. If we use these graphs to illustrate the eruption of the permanent teeth, it seems permissible to assume a high variability in the eruption sequence. This is in fair accordance with clinical experience. Nevertheless it seems necessary to establish the sequence of eruption as a standard.

For this reason, the eruption stage of each two pairs of teeth has been compared without regard to the chronological age of the persons involved. This method permits conclusions to be drawn as to the probability of a standard sequence of eruption for two pairs of specified teeth. The method is thoroughly described in the text. It should be mentioned that this method has been successfully used by Adler [1951] in demonstrating the effects of the continuous use of fluoride water on the eruption of the permanent teeth.

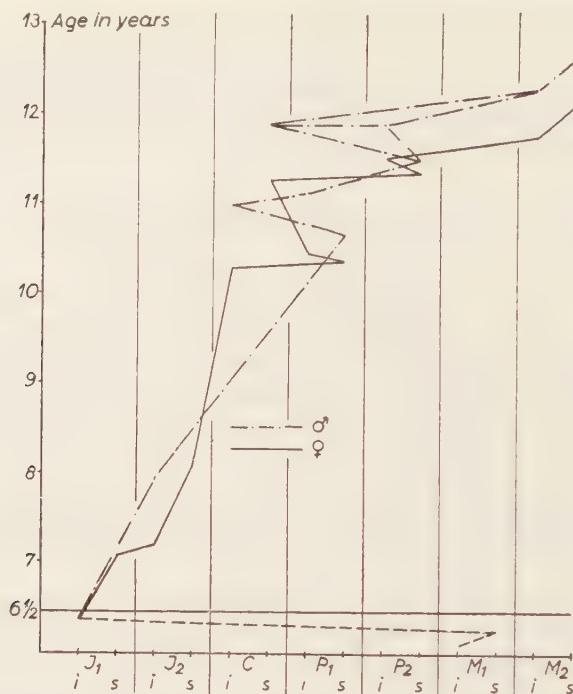


Fig. 1. The sequence of permanent tooth eruption in girls and in boys with regard to the mean eruption age of the individual teeth. The mean ages at which the specified teeth erupt, seem to be chronologically connected. The data compiled do not afford any evidence as to the sequence of eruption of the first molars and lower central incisors, in relation to the mean eruption age. This part of the curves is based on other evidence (mutual relationship of eruption of the individual teeth) and is separated from the main part by a horizontal line. This part of the curves is given in broken line.

Results. The sequence of tooth eruption is shown in fig. 1 for girls and boys in our main group, consisting of 8333 children of both sexes totally. As regards the mean age at the eruption of the first molars and of the lower median incisors, our data do not permit any conclusion. For all other teeth the eruption appears to come earlier in girls. A temporal difference between girls and boys in regard to the eruption of the permanent teeth is a well-known fact, frequently recorded in dental literature. However, besides this temporal differ-

ence, a slight difference in the eruption sequence between boys and girls is also obvious that has not been noticed before¹⁾.

In girls the lower cuspid erupts after the upper second incisors, while in boys the eruption of the upper first premolar occurs prior to that of the lower cuspid. Thereafter the eruption of the lower first premolar occurs similarly in boys and girls. Some differences do exist between boys and girls as regards the sequence of the eruption of the upper and lower second premolars and the upper canines too, while in both sexes the last two permanent teeth to erupt are the lower and upper second molars, disregarding the wisdom-teeth.

We are unaware of anybody having shown a sexuadimorphic difference in regard to the eruption of the permanent teeth. This sexuadimorphism of the eruption patterns holds true for our group as a whole and for most communities, the data of which are combined in our total group, but not for all ones! Though our data refer to a group large enough to permit definite conclusions, it seemed desirable to examine the sequence of eruption in other regions of Hungary than the lowland and in other parts of the world. Since in different populations there are only slight differences as regards the mean age at the eruption of the individual teeth (and these differences are apparently without any significance as regards the problem discussed), it seems preferable when comparing the data to confine ourselves to statements of the sequence of eruption in the different populations. The data are compiled in table 1.

When discussing the data it seems preferable to consider the different races and populations living under different conditions separately. Among European whites, three groups of Hungarians and four of other peoples are compared. In regard to boys, in all these seven groups alike, the upper first premolar is the first permanent tooth that erupts after the first molars and the first and second incisors; thus it is the seventh permanent tooth that erupts. The lower canine is in four groups the eighth tooth in the sequence, in two groups the ninth and in one the tenth. In girls, on the other hand, the lower canine is the seventh permanent tooth to erupt in five

¹⁾ While our report was at the printer, we received a chart by Dr. V. O. Hurme, Forsyth Dental Infirmary, Boston, Mass., U. S. A., showing the mean ages of tooth emergence with plus and minus limits of the standard deviation, prepared by condensing all accessible data of the literature on this topic. In this the reversal of the eruption sequence of these two specified teeth is obvious too, and it was noticed by Dr. Hurme. According to a paper by Dr. Hurme this fact has been known and caused much discussion among anthropologists.

Table I. The Sequence of Permanent Tooth Eruption in Boys and Girls
of Different Populations.

Population and sex	The sequence of the eruption of the permanent teeth											
	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
<i>A. European Whites:</i>												
Hungary, 8333 <i>Gödöny,</i> of 10 communities	B. G.	?	?	?	I _{1s}	I _{2i}	I _{2s}	C _i	P _{1s}	C _i	P _{2s}	C _s = P _{2i}
Hungary, 1083 Gönc and 6 adjacent comm.	B. G.	?	?	I _{1i}	I _{1s}	I _{2i}	I _{2s}	C _i	P _{1s} = P _{1i}	C _i	P _{2s}	P _{2i}
Hungary, 803 Szekszárd	B. G.	?	?	?	I _{1s}	I _{2i}	I _{2s}	C _i	P _{1s}	C _i	P _{2s}	C _s
Finnland, <i>Wuorinen,</i> 7155	B. G.	M _{1i}	I _{1i}	M _{1s}	I _{1s}	I _{2i}	I _{2s}	C _i	P _{1s}	C _i	P _{2s}	C _s
Sweden, <i>Dahlberg and Maunsbach,</i> 6030	B. G.	?	?	?	I _{1s}	I _{2i}	I _{2s}	C _i	P _{1s}	C _i	P _{2i}	P _{2s}
England, Frodsham, <i>Stones and al.,</i> 3294)	B. G.	M _{1s}	I _{1i}	M _{1i}	I _{1s}	I _{2i}	I _{2s}	P _{1s}	P _{2s}	P _{1i}	P _{2i}	C _s
Germany and Cent. Europe, <i>Röse,</i> 41021	B. G.	M _{1i}	M _{1s}	I _{1i}	I _{1s}	I _{2i}	I _{2s}	P _{1s}	C _i	P _{1i}	P _{2s}	P _{2i} = C _s
<i>B. American Whites:</i>												
Hagerstown, 471	B.											P _{1s} = C _i

New York, Hellman, 1042 ¹⁾	B. G.	I _{ii} M _{1i} M _{1s} I _{1s} I _{2i} I _{2s} C _i P _{1i} P _{1s} P _{2i} C _s P _{2s} P _{2i} M _{2i} M _{2s}	
Minneapolis, Cohen ²⁾	B. G.	M _{ii} = M _{1s} = I _{ii} M _{1s} I _{1s} I _{2i} I _{2s} C _i P _{1s} P _{1i} P _{2s} P _{1i} C _s P _{2s} C _s M _{2i} M _{2s}	
Holland, Mich., Steggerda and Hill ¹⁾	B. G.	M _{ii} = M _{1s} = I _{ii} M _{1s} I _{1s} I _{2i} I _{2s} C _i P _{1s} P _{1i} P _{2s} C _s P _{2i} M _{2i} M _{2s}	
Chinese, Sullivan, 1532	B. G.	M _{ii} M _{1s} I _{ii} I _{1s} I _{2i} I _{2s} P _{1s} P _{1i} C _i P _{2s} P _{2i} C _s M _{2i} M _{2s}	
Zulus, Suk, 1008	B. G.	M _{ii} M _{1s} I _{ii} I _{1s} I _{2i} I _{2s} C _i P _{1s} P _{1i} C _s P _{2s} P _{2i} M _{2i} M _{2s}	
American Negr. Steggerda and Hill ¹⁾	B. G.	M _{ii} = I _{ii} M _{1s} I _{1s} I _{2i} I _{2s} C _i P _{1s} P _{1i} C _s P _{2s} M _{2i} M _{2s}	
Maya, Steggerda and Hill ¹⁾	B. G.	M _{ii} M _{1s} I _{ii} I _{2i} I _{1s} I _{2s} P _{1s} P _{1i} C _i P _{2s} C _s M _{2i} P _{2i} M _{2s}	
Navajo, Steggerda and Hill ¹⁾	B. G.	M _{ii} M _{1s} I _{ii} M _{1s} I _{2i} I _{1s} I _{2s} C _i P _{1s} P _{1i} C _s P _{2s} C _s P _{2i} M _{2i} P _{2s} = M _{2s}	

¹⁾ Results of serial studies on the same individuals over longer periods.²⁾ No further data are accessible to us.

Signs used: B: boys; G: girls; I: incisors; C: canines; P: premolars; M: molars. 1: first of the specified tooth type; 2: second of the specified tooth type; s: upper tooth; i: lower tooth; =: eruption of two or more teeth at the same age.

Where the sequence of eruption is identical in boys and girls, data are written in the mid-line.

groups; in these it thus erupts prior to the first upper premolar. In the other two groups it is the eighth tooth to erupt, being preceded by the upper first premolar. Among American whites the first upper premolar is in boys the seventh permanent tooth that erupts in three out of four groups; in the fourth it is the lower canine. In girls, on the other hand, the lower canine is the seventh tooth that erupts in all four groups.

Less uniformity is seen in the data of coloured races. In two populations (Chinese, Maya) no difference is found between girls and boys as regards the mean eruption age of the two specified teeth, the first upper premolar being in both populations the seventh, the lower canine the ninth permanent tooth that erupts. In one group the lower canine erupts prior to all other teeth in boys and girls alike (Zulu). In American negroes and Navajos, however, a similar change of the sequence is seen as was demonstrated in most groups of whites: in boys, the upper first premolar is the seventh permanent tooth in the sequence of eruption, the lower cuspid the ninth, while in girls the lower canine erupts prior to the upper first premolar.

Summarizing the data it can be stated that in eight population groups of whites (out of a total of eleven) and in one group of coloured races living under conditions similar to the poorest classes of whites (American negroes) a sexualdimorphic pattern in the eruption sequence of the upper first premolar and the lower canine was demonstrated. One group of whites is *a priori* unsuitable to demonstrate any difference of this kind, the lower canine erupting prior to all other teeth of the second series of erupting permanent teeth even in boys (New York). In one group (Frodsham) there is a marked difference between girls and boys, the eruption of the lower cuspid occurring in boys as the tenth permanent tooth, being preceded by the upper second and also the lower first premolar, while in girls it erupts as the eighth tooth, prior to the aforementioned premolar teeth. There is only one group (mixed Central-European population, compiled by Röse more than fifty years ago) where such a difference does not exist (in spite of it being possible). Thus these data indicate that a sexualdimorphic difference exists in the eruption sequence in regard to the upper first premolar and the lower canine in whites, being characterized by an earlier eruption of the lower canine in girls. This sexualdimorphism of the pattern is, however, apparently not constant.

As regards the reason for such inconstancies, the variability of the mean age at the eruption should be taken into consideration

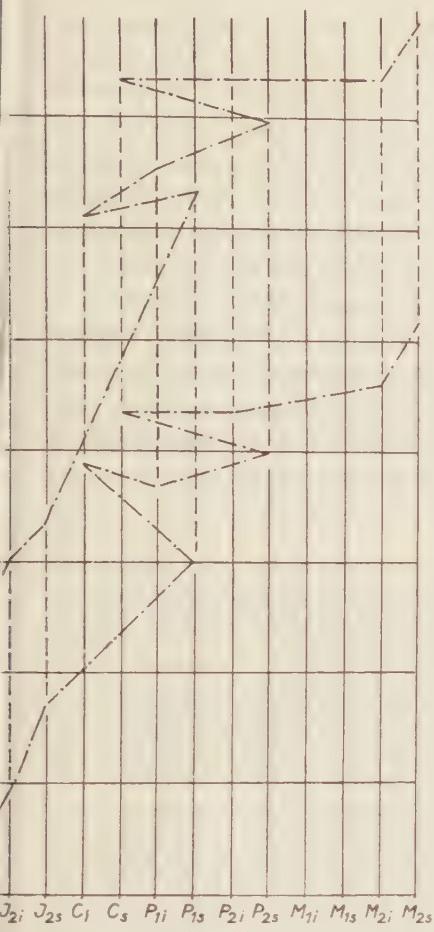


Fig. 2.

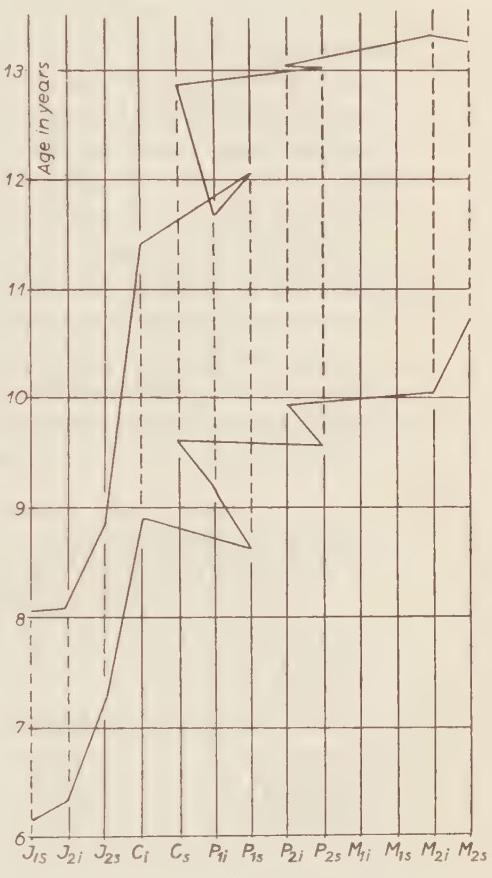


Fig. 3.

Fig. 2. Variations in the mean eruption time of the permanent teeth in boys, taking into consideration variations of $\pm \sigma$. Maximum and minimum ages are chronologically connected similarly to fig. 1. Interrupted vertical line: variability of mean eruption time. *Fig. 3.* Variations in the mean eruption time of the permanent teeth in girls. Construction and explanation identical with fig. 2.

first of all. In figs. 2 and 3 the variability in the eruption sequence is demonstrated in boys and girls of our main group, taking into consideration a variation in the range of $\pm \sigma$ from the mean. Regarding these graphs one becomes aware of the uselessness of employing mean data for tooth eruption. However, a certain sexual dimorphic trend of the eruption sequence is obvious in these graphs too.

A further fact has to be taken into consideration also. It has been pointed out by Steggerda and Hill [1942] that the temporal difference between boys and girls as regards the eruption, though demonstrated for all permanent teeth, is most marked for the canines. The earlier eruption of the lower canine in girls compared with boys may become manifest in a change of the eruption sequence between the upper first premolar and this tooth, if *a priori* there is no greater difference in the mean age at the eruption of these two types of teeth than the relative priority of the lower canine compared with the upper first premolar. Whether or not a change in the pattern becomes manifest, thus depends on the temporal difference existing between the mean ages at the eruption of these two types of teeth in boys. If we take this item into consideration in regard to the white and coloured groups for which a reversal of the eruption sequence was not demonstrated, the following differences are recorded:

Table 2. The Difference of the Mean Eruption Time of the Upper First Premolar and the Lower Canine in Boys and Girls in those Populations where no Reversal of the Eruption Sequence Occurs.

Population and reference	Eruption of the upper first premolar occurs prior to the lower canine by years	
	in boys	in girls
Frodsham/Stones	0.48	0.20
Germany/Röse	0.75	0.17
New York/Hellman	-0.13	-0.57
Chinese/Sullivan	0.71	0.57
Zulu/Suk	-0.48	-0.64
Maya/Steggerda and Hill.	0.90	0.30

The data in the table clearly demonstrate a relative priority of the eruption of the lower cuspid in girls in comparison with the upper first premolars and with similar data for boys.

It is interesting to note that we were unable to detect a similar regular change in the eruption sequence of the upper cuspids for both girls and boys, though it should be expected according to the data of Steggerda and Hill [1942].

The statistical significance of this sexualdimorphic difference should be clarified still. For this purpose a special method has been

used that was devised (on our behalf) by Dr. A. Rényi, director of the Institute of Applied Mathematics of the Hungarian Academy of Sciences.

With this method two pairs of teeth are compared as regards the stage of eruption. The method is demonstrated, e.g. by comparing the eruption stage of the upper first premolars and the lower canines in boys and in girls. The chronological age of the subjects is not taken into consideration at all.

Nothing can be said about the stage of eruption of these two pairs of teeth in subjects in whom none is erupted if statements are based upon clinical examinations, excluding x-rays. Neither can anything be said in regard to persons in whom both pairs are already erupted, since we are unable to state in what sequence the eruption occurred. Thus only those persons are to be considered in whom these tooth pairs have reached other eruption stages than the aforementioned two ones. There are seven possibilities to be considered:

1. persons with 2 upper premolars and 0 lower canine;
2. persons with 2 upper premolars and 1 lower canine;
3. persons with 1 upper premolar and 0 lower canine;
4. persons with 1 upper premolar and 1 lower canine;
5. persons with 1 upper premolar and 2 lower canines;
6. persons with 0 upper premolar and 1 lower canine;
7. persons with 0 upper premolar and 2 lower canines.

All subjects falling in any of these seven groups are to be counted. Knowing the number of subjects in each group, an eventual surplus of the upper first premolars or lower canines for the total group is easily computed. This is to be divided by the number of subjects in all seven groups. A quotient of more than 0,80 is highly significant; quotients of 0,70 are on the borderline of significance.

Using this special method, the eruption of these two specified teeth was determined in girls and in boys in three communities, forming parts of the 10 comprised in our main group. Additionally, a similar grouping was performed of the subjects in the school population of Gönc and of the adjacent six smaller villages. The results are graphically recorded in fig. 4 a-d, which show the distribution of boys and girls in these seven groups. A sexual dimorphic pattern is obvious. While in boys the surplus of premolars is preponderant, in girls rather a surplus of lower canines exists. The data of the three communities and of the Gönc-district are summarized in table 3.

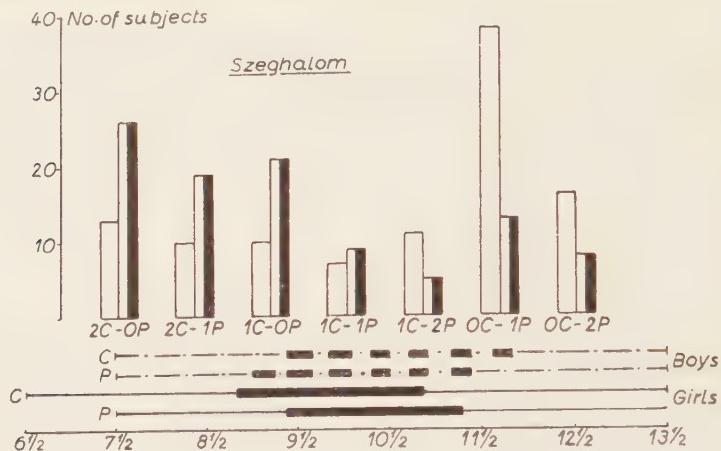


Fig. 4 a.

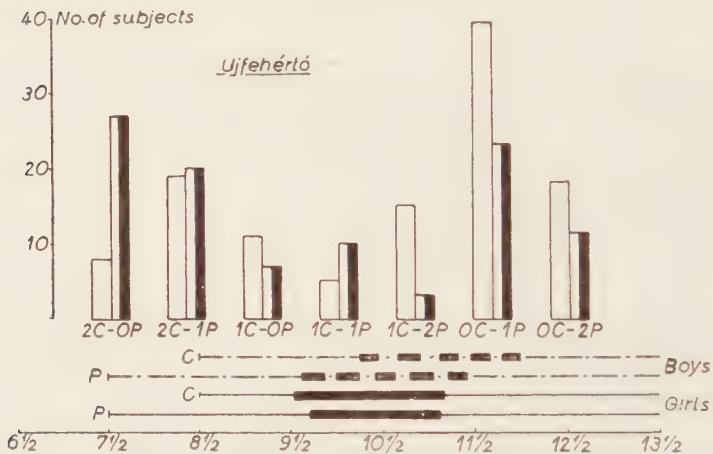


Fig. 4 b.

Fig. 4. Distribution of the different eruption stages of upper first premolars and lower canines in boys (white columns) and girls (half-black columns), a = at Szeghalom, b = at Ujfehértó, c = at Dévaványa, d = at Gönc and in adjacent 6 small villages. In the horizontal lines below the columns the temporal variations in the eruption of these specified teeth are shown. Thick lines demonstrate the interval between the 25 and 75 per cent presence stages; thin lines between 0 and 100 per cent presence. Ages in years.

While in boys there is a clear and marked surplus of upper premolar teeth in the final sum in all four groups, in girls a surplus of the lower canine is demonstrated only at Szeghalom and Ujfehértó. At Dévaványa and in the district of Gönc upper premolars form

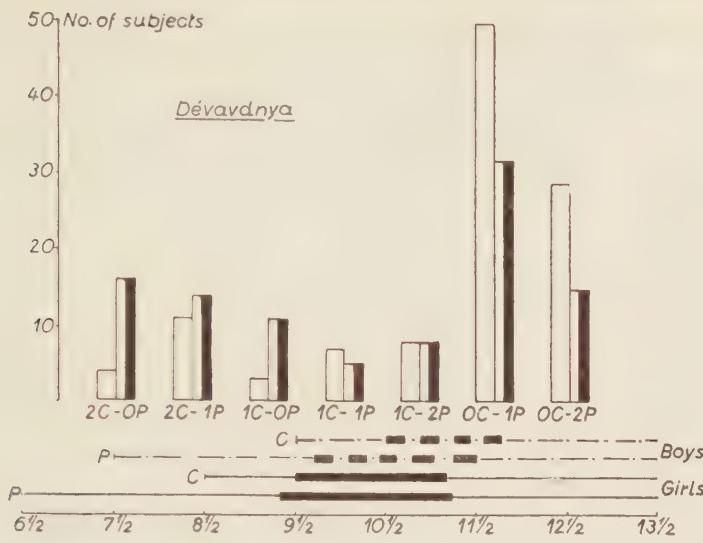


Fig. 4.c.

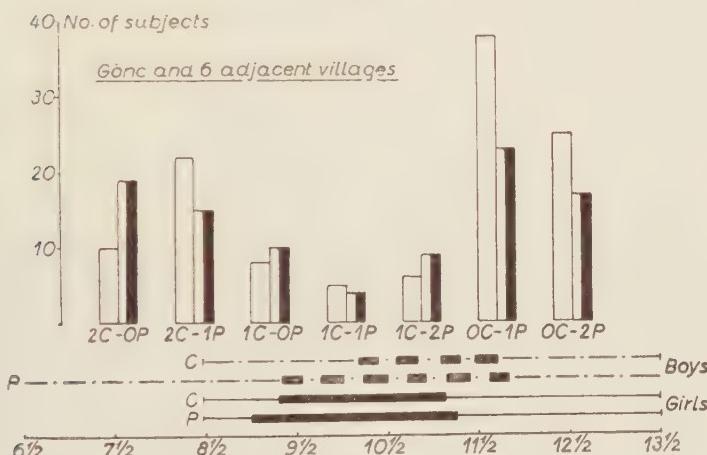


Fig. 4.d.

a slight surplus even in girls, in spite of the mean age at the eruption of the lower canine occurring prior to that of the upper first premolar tooth. This seems to be contradictory; however, it should be due to those few subjects in whom by accidental reasons an earlier eruption of the upper premolar occurs. In figs. 4 a-d the temporal variations in the eruption of these two pairs of teeth are shown also. The figures demonstrate that in some subjects eruption occurs outside the

Table 3. The Mutual Stage of Eruption of Lower Canines and Upper First Premolars in Boys and Girls in Different Regions of Hungary.

Stage of eruption	Number of subjects displaying specified stages of mutual eruption in										Total
	Szeghalom		Ujfehértó		Dévaványa		Gönc-distr.				
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	
2 C - 0 P	13	26	8	27	4	16	10	19	35	88	
2 C - 1 P	10	19	19	20	11	14	22	16	62	69	
1 C - 0 P	10	21	11	7	3	11	8	10	32	49	
1 C - 1 P	7	9	5	10	7	5	5	4	24	28	
1 C - 2 P	11	5	15	3	8	8	6	9	40	25	
0 C - 1 P	38	23	39	23	50	32	38	23	165	101	
0 C - 2 P	16	8	18	11	29	15	25	17	88	51	
Total number of subjects	105	111	115	101	112	101	114	98	446	411	
Total surplus of C	—35	48	—44	33	—94	—13	—33	—2	—217	66	
Surplus of C per subject $\times 100$	—33	43	—38	32	—84	—13	—39	—2	—49	15	
Difference between boys and girls per subject		0.76		0.70		0.71		0.37		0.64	

25 and 75 per cent limits. In fig. 5 the original curves for the school population of the Gönc district are reproduced; it is quite obvious that though the eruption of the upper first premolar as a rule occurs later than that of the lower canine in girls, its eruption starts in some persons prior to that of the lower canine. These exceptions finally cause a difference to exist with a slight surplus of upper first premolars even in girls. Taking into consideration the difference between boys and girls in regard to the eruption stages, a borderline significance was found at Szeghalom, Ujfehértó and Dévaványa, while the difference in the Gönc district was not significant though marked. Summarizing all data, the difference is just below the significance level.

In the initial stages of our caries research unfortunately no attention was paid to this peculiar problem. In this respect we are thus unable to study the school population of all 10 communities, composing our total group of 8333 children.

However, this method seems suitable to establish the sequence of tooth eruption in spite of great temporal variations in regard

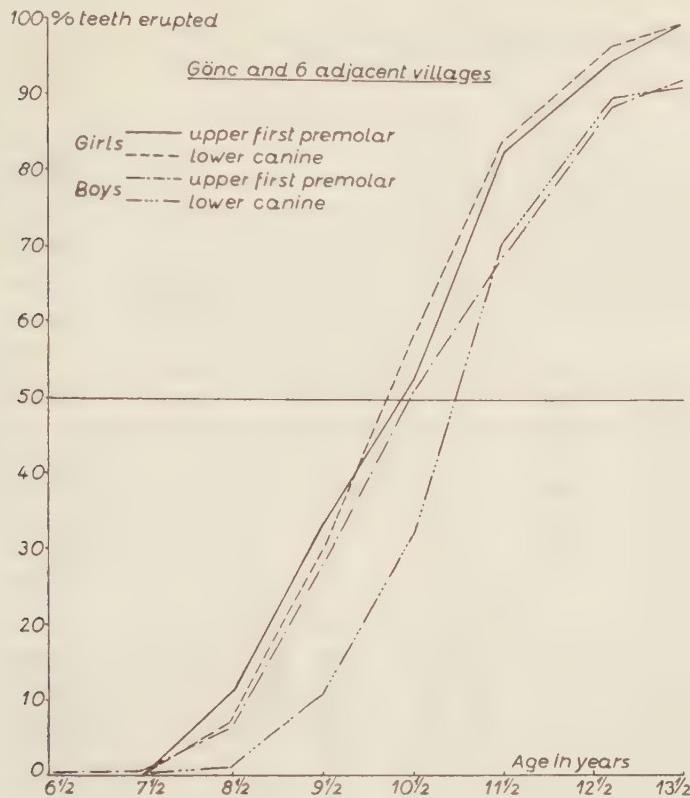


Fig. 5. Accumulated presence curve of upper first premolars and lower canines in boys and in girls of Gönc and adjacent 6 villages. It is obvious that even in girls the upper first premolar starts in a few persons to erupt earlier than the lower canine, whereas the 50 per cent presence of the canine is earlier attained than that of the premolar.

to the age at the eruption of identical teeth in different individuals. This variability has been demonstrated in figs. 2 and 3. The question now is whether the variations shown in the figures are due to the fact that the population of our 10 communities is composed of groups of persons in whom all teeth erupt early, at ages near the mean eruption age for the individual teeth, or late, or whether they are due to variations in the sequence of eruption of the different teeth in different individuals. Klein and co-workers [1937] suppose that one should be able to distinguish between "early" and "late" eruptors on the basis of the eruption data of one specified type of teeth. In

Table 4. Comparison of the Mutual Stage of Eruption of Two Pairs of Specified Teeth in Boys and Girls.

Teeth compared	Number of comparable subjects	Boys		Girls	
		Surplus of first mentioned tooth total	in 1 pers.	Surplus of first mentioned tooth total	in 1 pers.
I _{1sup} : I _{1inf}	254	—437	—1.72	214	—355
I _{2sup} : I _{2inf}	272	—413	—1.52	259	—363
C _{sup} : C _{inf}	339	—407	—1.20	443	—636
P _{1sup} : P _{1inf}	458	293	+0.64	399	137
P _{2sup} : P _{2inf}	432	166	+0.38	379	62
M _{1sup} : M _{1inf}	60	— 17	—0.28	46	— 36
M _{2sup} : M _{2inf}	239	—236	—0.99	285	—323

*A. Comparison of upper teeth with lower teeth
(Upper and lower mates):*

I _{1sup} : I _{1inf}	254	—437	—1.72	214	—355	—1.66
I _{2sup} : I _{2inf}	272	—413	—1.52	259	—363	—1.40
C _{sup} : C _{inf}	339	—407	—1.20	443	—636	—1.42
P _{1sup} : P _{1inf}	458	293	+0.64	399	137	+0.34
P _{2sup} : P _{2inf}	432	166	+0.38	379	62	+0.16
M _{1sup} : M _{1inf}	60	— 17	—0.28	46	— 36	—0.78
M _{2sup} : M _{2inf}	239	—236	—0.99	285	—323	—1.13

B. Comparison between different upper teeth:

M ₁ : I ₁	332	567	1.71	246	425	1.73
I ₂ : P ₁	814	1238	1.52	814	1274	1.57
P ₁ : C	610	765	1.25	496	604	1.22
P ₁ : P ₂	514	496	0.96	523	588	1.12
C : P ₂	422	—227	—0.54	360	— 53	—0.15
C : M ₂	341	251	0.72	381	382	1.00
P ₂ : M ₂	499	523	1.05	462	504	1.10

C. Comparison between different lower teeth:

M ₁ : I ₁	110	143	1.30	75	104	1.39
C : P ₁	285	90	0.32	336	255	0.76
P ₁ : P ₂	412	407	0.99	472	511	1.08
C : P ₂	412	468	1.14	533	713	1.34
P ₂ : M ₂	313	— 23	—0.08	394	52	0.13

*D. Comparison between specified upper and lower teeth
(different ones):*

M _{1sup} : I _{1inf}	121	108	0.89	85	83	0.98
I _{1sup} : I _{2inf}	192	164	0.85	164	161	0.98
C _{sup} : P _{2inf}	356	— 28	—0.08	369	29	0.08
C _{sup} : M _{2inf}	317	— 27	—0.09	345	53	0.15
P _{1sup} : C _{inf}	446	217	0.49	411	— 66	—0.15

order to elucidate this question it seemed necessary to us to carry out comparisons similar to those made between the upper first premolar and the lower canine between other teeth also. Such a study was carried out in the population of the small transdanubian town Szekszárd by Adler, but the scope was at that time restricted to investigate the mutual eruption stage of the lateral teeth. No distinction was made between males and females. With the aid of this method it was demonstrated by Adler in a second study that premolars erupt earlier in fluorine-free regions compared with regions using naturally fluorinated domestic water.

Comparisons have been made between the populations of Szeghalom, Ujfehérváros, Dévaványa and Gönc and those of the adjacent six smaller villages. The final results are shown in summarized form in table 4. A few individual data of certain patterns are demonstrated in fig. 6.

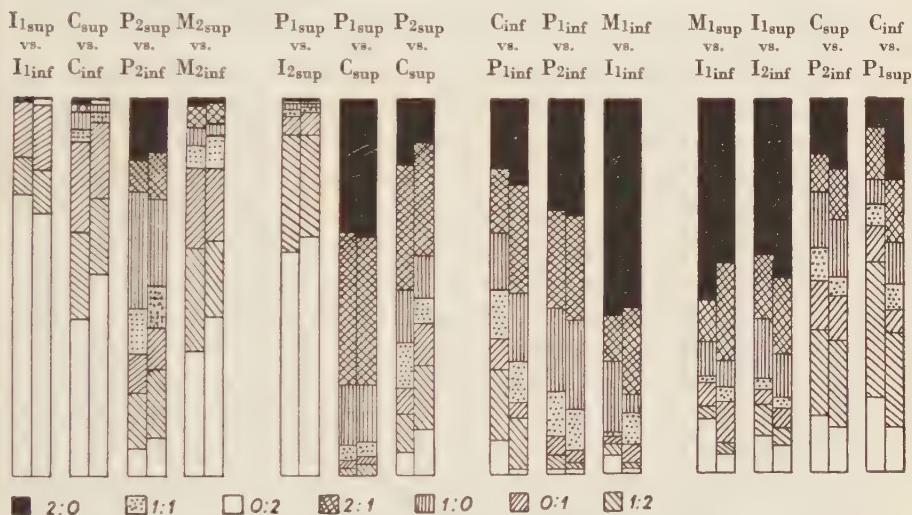


Fig. 6. Histograms on the mutual eruption stage of two pairs of specified teeth—selected instances, on percentage basis. The specified teeth compared are shown on the heads of the columns. The proportion 2:0, 2:1, 1:0 etc. indicate the relation of the tooth mentioned in the upper row versus the one mentioned in the lower row. The most marked sexual dimorphism is obvious in the last vertical column (upper premolar and lower canine). Preponderance of the one tooth over the other is most marked in the comparison of upper and lower central incisors, lower first molar and lower central incisor, upper first molar and lower central incisor, upper first premolar and upper lateral incisor. No preponderance of the one tooth over the other is obvious in comparing upper and lower second premolars, upper canine and lower second premolar, etc. Left columns: boys; right columns: girls.

The data compiled in table 4 and partly shown in fig. 6 permit the following conclusions:

A) When comparing corresponding teeth in the upper and lower jaws it is found that the teeth in the lower jaw usually erupt earlier than those in the upper jaw. The difference is significant except for the first molars in boys. Another exception are the premolars: usually those in the upper jaw erupt prior to their lower mates. The difference in boys in regard to the first premolars is nearly on the borderline of significance, while the difference in boys in regard to the eruption of the second premolars and in girls of the first and second ones is not significant. As regards the data for first molars no great importance should be attributed to them, since in our material only very few persons were found in whom the eruption data of the first molars could be compared. This is due to the fact that in Hungary the children begin school after the 6th birthday. The restricted validity of our data is obvious also from Gödény's [1951] first report in this series.

B) As regards the sequence of eruption in the maxilla our data contradict the views expressed by Schultz [1950] and by Broom and Robinson [1951], according to whose opinions the central incisors erupt prior to the first molars. In our material, in boys as well as in girls, there is a very marked preponderance of first molars in comparison with central incisors. A second problem of interest was to examine the mutual relation between the tooth that is the latest to erupt in the first group of permanent teeth (i.e. the lateral incisor) and the earliest tooth of the second group (i.e. the first permanent premolar). There is a very marked, highly significant difference in favour of the second permanent incisor, indicating that there really is a time-lag between the two stages of permanent tooth eruption, the first molars and all incisors belonging to the first one, and all other permanent teeth—not including the third molars—to the second. A slight difference seems to exist between boys and girls in regard to the eruption of the upper canine, compared with the second premolar and the second molar, but quantitatively this sexual-dimorphic difference is far from being significant.

C) As regards the mutual eruption stage of the central incisors and the first molars, a temporal advance of the molar was found in the mandible also as was the case in the maxilla. Compared with the first premolar the lower canine erupts markedly earlier in girls than in boys; the difference, however, is not statistically significant.

Similarly, no statistical significance can be established for the difference between boys and girls in regard to the mutual stage of eruption of the canine and the second premolar, and of the second premolar and the second molar. Thus the sequence of eruption of the mandibular teeth is as follows: $M_1, I_1, I_2, C, P_1, P_2$ or M_2 .

D) The few comparisons between different upper and lower teeth demonstrate that even the upper first molar erupts prior to the lower central incisor; similarly the upper central incisor erupts prior to the lateral lower one. There seems to be a slight sexual-dimorphic trend in the mutual behaviour of the upper canine and the lower second premolar or lower second molar but without statistical significance. It was hoped that a comparison between the upper canine and the lower second premolar would throw some light upon the frequency of inclusion of these two types of most frequently included teeth. However, the differences actually found do not permit any conclusion.

24 pairs of teeth were compared and in no other instance were we able to find a similar sexualdimorphic difference as has been demonstrated between the upper first premolar and the lower canine. As to the cause of this sexualdimorphism we are unable to make any definite statement. It is a wellknown fact that in girls tooth eruption occurs at an earlier age than in boys; however this fact offers no explanation of the observed difference in the sequence of eruption. Though the data compiled in table 4 permit no definite conclusions, at least some conjectures as to the manner of producing the sexual-dimorphic tooth eruption pattern can be made:

- a) There is a slight difference between boys and girls in regard to the mutual eruption stage of the upper and lower first premolars, the eruption of the upper premolars compared with that of the lower ones occurring earlier in boys than in girls.
- b) The reverse holds true as regards the eruption of the canines.
- c) In the mandibular dental arch the canine of girls in relation to the first as well as to the second premolar seems to erupt earlier than is the case in boys.

The combined effect of the earlier eruption of the lower canine in girls and of the earlier eruption of the upper first premolar in boys is the observed sexualdimorphism in the eruption sequence of these two specified pairs of teeth. While these observations satisfactorily explain the manner in which the sexualdimorphic eruption pattern is produced, they do not give any hint as to the reasons for it. Hor-

monal influences can hardly play any role, since we cannot assume a hormonal influence that is restricted to specified types of teeth without having any effect on other teeth which erupt practically during an identical period of life.

Summary.

The sequence of tooth eruption of the permanent dentition of the normal school population in Hungary has been studied. A difference was found between girls and boys: after the first permanent molars and incisors usually the upper first premolars erupt in boys but the lower canines in girls. This sexuadimorphic pattern of permanent tooth eruption was demonstrated in most other populations by analysis of literature accessible to the authors. In a few populations no sexuadimorphism was found, but even in these there was a markedly earlier eruption in girls of the lower canine in relation to the upper first premolar. A method is described that permits statistical comparison of the mutual stages of eruption of two pairs of specified teeth. This method was used in comparing the eruption sequence of corresponding teeth in the upper and lower jaws, the eruption sequence in the upper and lower dental arches, and the sequence of different upper and lower teeth. A sexuadimorphic pattern could be demonstrated in regard to the upper first premolars and lower canines.

Résumé.

Etude de l'ordre d'éruption des dents de la seconde dentition chez des enfants dans des écoles normales de la Hongrie. Une différence fut établie entre des jeunes filles et des garçons en ce qui concerne l'ordre d'éruption. Description d'une méthode qui rend possible une comparaison statistique des phases mutuelles d'éruption pour deux paires de dents spécifiées. La méthode est employée pour comparer l'ordre d'éruption des dents correspondantes dans la mâchoire supérieure et inférieure, etc.

Zusammenfassung.

Die Reihenfolge des Zahndurchbruches für das Permanentzahngebiss ist bei normaler Schuljugend in Ungarn untersucht worden. Für Mädchen und Jungen ist ein Unterschied festgestellt worden:

Nach den ersten permanenten Molaren und Inzisoren pflegen gewöhnlich die ersten oberen Prämolaren bei den Jungen durchzubrechen, bei den Mädchen dagegen die unteren Kaninen. Dieses sexualdimorphe Schema des Permanentzahndurchbruches wurde durch Analysen von Literatur, welche den Verfassern zugänglich war, bei den verschiedensten anderen Populationen aufgezeigt. In einigen von ihnen wurde kein Sexualdimorphismus entdeckt, aber selbst bei diesen fand sich ein merkbar früherer Durchbruch des unteren Kaninus in bezug auf die obere erste Prämolare bei den Mädchen. Eine Methode, welche den statistischen Vergleich der beidseitigen Durchbruchsstadien von zwei Paaren angegebener Zähne ermöglicht, wird beschrieben. Diese Methode wurde angewendet beim Vergleich der Durchbruchsfolge einander entsprechender Zähne im Ober- und Unterkiefer, der Durchbruchsfolge in den oberen und unteren Dentalbögen und der Aufeinanderfolge von verschiedenen oberen und unteren Zähnen. Ein sexualdimorphisches Schema ließ sich aufstellen im Hinblick auf die oberen ersten Prämolaren und die unteren Kaninen.

REFERENCES

- Adler, P.: Az oldalsó fogak váltódásának sor- és időrendje. Fogorv. Szle., in press (Hung.). – Id.: Acta Med. Hung. 2, 349, 1951. – Broom, R. and J. T. Robinson: Nature 167, March 17, 1951. – Dahlberg, G. and A. B. Maunsbach: Acta Genet. a. Stat. Med. 1, 77, 1948. – Gödény, E.: Acta Genet. et Stat. Med. 2, 331, 1951. – Hellman, M.: Am. J. Orthod. a. Or. Surg. 29, 507, 1943. – Klein, H., C. E. Palmer and M. Kramer: Growth 1, 385, 1937. – Schultz, A. H., quoted by Broom and Robinson, from Proc. Am. Phil. Soc., Oct. 1950. – Steggerda, M. and Th. J. Hill: Am. J. Orthod. a. Or. Surg. 28, 361, 1942. – Stones, H. H., F. E. Lawton, E. R. Bransby and H. O. Hartley: Brit. dent. J. 90, 1, 1951.

From the State Institute of Human Genetics and Race Biology
(Head: Professor Gunnar Dahlberg, Uppsala, Sweden)

MENARCHE IN SCHOOLGIRLS

Effects of Joint and of Separate Education

By TORSTEN ROMANUS

The incentive to the present investigation was a speech read before the 15th Congress of Northern Educators at Stockholm in August 1948 by Professor A. Salo and entitled: "The Effects of Coeducation and of Separate Schooling on Girls' Development". Salo had found that in Finland girls from coeducational schools have their menarche on an average 7.5 months earlier than those from schools exclusively for girls. Professor Salo has very kindly let me know that the investigation was based on a series of 2,086 pupils from girls' schools with a mean age at menarche of 14.13 years and on 1,544 girls from coeducational schools whose average age at menarche was 13.5 years. (The investigation has been published in Finnish only.) For various reasons, among others the proposed reformation of the Swedish educational system, it might be interesting to see how Salo's results would apply in Sweden.

Here it may be mentioned already that the primary aim of the present investigation was to compare the age at the first menstrual period of girls from coeducational schools with the corresponding age for girls in girls' schools. The essential thing is therefore that the two groups must be comparable, which means that their selection and composition must be exactly alike.

Prior studies on menarche have had very variable results. The mean age differs markedly in different investigations. This will be seen in the appended tables giving some examples from different countries and periods. It has in this connection been claimed that menarche varies with the climate and has come progressively earlier in recent generations. These investigations have been based either on data from sexually mature women who have been more or less

Table 1. The mean age at menarche as given by different authors.

		Year	Number	Menarche age at
Krieger	Germany . . .	1869	6546	16.35
Schlichting	Munich	1880	8881	16.57
Engelman	North America .	1902	12402	14.49
Essen-Möller	Sweden	1906	5000	15.72
Schaeffer	Berlin	1908	10500	16.22
Yamasaki	Japan	1909	4861	14.92
Sanes	Pittsburg	1916	2864	14.58
Malmio	Finland	1919	1219	15.56
Curjel	India	1920	489	13.63
Heyn	Schlesien	1920	2705	15.86
Bolk	Holland	1923	1800	14.06
Kennedy	Edinburgh . . .	1933	10219	15.04
Engle and Shelesnyak .	New York . . .	1934	250	13.53 (jewesses)
Skerlj	Norway	1939	4050	15.09
Frommolt	Sweden	1936	5548	15.02-16.20
Breipohl	Königsberg . . .	1937	284	13.24 (school girls)
Gini and Orchì	Italy	1939	15202	14.41 (multiparae)
Lennér	Sweden	1940	2000	14.49
Samuelsson	Sweden	1942	3014	15.00
v. Castricum	South Africa . .	1946	2233	14.65

grown up, or on information from fairly old schoolgirls. The difference in composition of the series explains the variability of the results. The possible sources of error will be discussed further on.

Materials and Methods.

The basic data for the investigation were gathered by having pupils at the respective schools fill out questionnaires which were handed out and collected by a suitable member of the teaching staff, usually the physical training mistress.

The following points were covered by the questionnaire: Type of school (coeducational or girls') and where located; the pupil's initials, date of birth, class in school, father's employment, number of terms in present school, date of first period (year and month), type of school in the year preceding the first menstrual period (1. coeducational school, 2. girls' school, 3. elementary school with mixed classes, 4. elementary school with separate girls' classes), date when filling out form, chronic illnesses such as heart disease, tuberculosis etc., and whether menstruation had not commenced.

Table 2. Classification of the cases.

Town	When questioned pupil at						Girls' school			
	Coeducational school			Excluded for			Excluded for			
	No. of returns	Time for menarche not given	Not menstruated yet	Other reasons	No. analyzed	No. of returns	Time for menarche not given	Not menstruated yet	Other reasons	No. analyzed
Luleå	30	3	—	—	27	28	1	1	—	26
Umeå	72	—	—	—	72	52	1	—	4	47
Östersund	49	4	—	1	44	77	5	1	2	69
Härnösand	49	—	—	2	47	50	2	—	—	48
Sundsvall	63	5	—	3	55	70	3	—	—	67
Gävle	47	3	—	—	44	42	—	1	—	41
Falun	49	—	—	4	45	44	1	—	1	42
Uppsala	201	15	—	8	178	198	16	1	1	180
Västerås	14	3	—	—	11	90	5	1	—	84
Karlstad	95	2	—	5	88	141	18	1	3	119
Eskilstuna	41	2	—	—	39	57	1	1	—	55
Kristinehamn	31	2	—	—	29	24	—	—	15	9
Stockholm	116	22	1	4	89	322	16	—	9	297
Södertälje	33	1	—	—	32	49	1	1	—	47
Nyköping	27	—	—	—	27	55	3	2	—	50
Linlöping	41	2	—	—	39	81	1	—	2	78
Norrköping	79	3	—	—	76	150	38	3	—	109
Skövde	40	22	—	2	16	53	11	1	1	40
Skara	70	2	—	—	68	34	12	1	—	21
Vänersborg	65	12	—	1	52	64	3	—	—	61
Uddevalla	44	7	—	—	37	40	7	1	1	31
Jönköping	69	17	—	1	51	90	1	—	—	89
Borås	70	4	—	—	66	—	—	—	—	—
Göteborg	51	2	—	—	49	129	—	—	3	126
Växjö	88	—	—	—	88	89	—	—	2	87
Halmstad	93	—	2	2	89	77	5	1	3	68
Kalmar	74	4	—	—	70	80	4	1	1	74
Karlskrona	72	1	1	3	67	104	1	1	1	101
Kristianstad	58	2	—	2	54	44	9	—	—	35
Landskrona	22	3	—	2	17	63	7	—	—	56
Lund	—	—	—	—	—	60	—	1	—	59
Total	1853	143	4	40 ¹⁾	1666	2457	172	20	49 ²⁾	2216
Per cent.	100.0	7.7	0.2	2.1	90.0	100.0	7.0	0.8	2.0	90.2

¹⁾ Date of birth faulty or not given: 27; not at school year preceding menarche: 7; school during year before menarche not specified: 5; in sanatorium at time of menarche: 1.

²⁾ Date of birth faulty or not given: 18; not at school year preceding menarche: 3; school during year before menarche not specified: 28.

The form was distributed among pupils in the three highest classes at the schools selected. The physical training mistress was usually appointed for the job as she would be most likely to know whether the pupils had started menstruating or not. The schools were selected in pairs from the same town, one being a communal girls' school and the other a coeducational upper section of a secondary school. The pupils had thus been able to choose between coeducational and separate instruction. All parts of the country are represented, from Luleå in the north to Lund in the south.

Ages are given in decimal notation in the tables, 0.1 years representing 1.2 months, 0.2 years 2.4 months, etc.

Returns came in from altogether 4,310 girls, among whom 1,853 attended coeducational schools when the form was filled out and 2,457 were in girls' schools.

Sources of Error.

To begin with it should be noted that there exists no sharp division between girls who menstruate and girls who do not. There are for example women who never do menstruate, although they probably are not very numerous. It is perhaps more likely that girls have a menstrual period once or twice and then do not menstruate regularly. In such cases the question arises whether they shall be considered to have entered menarche, and the variations are limitless. These cases are better understood now that we know more about the hormonal control of menstruation, and our resources for treating abnormalities are better too. According to our present knowledge the menstrual cycle is governed by hormonal action (oestrone and progesterone) on the uterine mucosa (*Dahlberg and Åkesson [1930]*). It would not be surprising, therefore, if studies on menarche included a group of doubtful cases. But such groups are seldom reported, in part surely because girls are reluctant to admit that they are not quite normal in that respect and therefore give erroneous or vague answers. Such erroneous answers obviously must affect the end results and may give rise to large or small deviations of the mean for the series. Yet it is hard to say in what direction this source of error will affect the means, since it is not easy to know to what extent and in what manner the data are incorrect. A girl who has not menstruated would probably be more likely to put her imaginary first period at a late than at an early date. If, on the other hand, she has menstruated once or twice she would, with some

justification, give that date as the onset of menstruation even if there has been no regularity and a relatively long intermission since then. At any rate we have nothing definite to go on in this respect.

Quite a few of the girls in this investigation did not complete their forms. It may be surmised that some, but only some, of these girls belong to the type with some menstrual abnormality (cf. table 3).

Table 3. Age distribution of the girls when questioned.

Age of the girls when questioned (in years)	Included in the investigation		Excluded ¹⁾	
	Number	%	Number	%
13 - 14	5	0.1	—	—
14 - 15	84	2.2	9	2.5
15 - 16	566	14.6	31	8.7
16 - 17	974	25.1	94	26.3
17 - 18	1166	30.0	94	26.3
18 - 19	691	17.8	84	23.5
19 - 20	298	7.7	39	10.9
20 -	98	2.5	6	1.7
Total	3882	100.0	357	100.0

¹⁾ Among the 428 excluded girls the age could not be computed for 48, and 23 stated they had not menstruated yet. These 71 are therefore not included in the table.

It turns out that the percentage of girls with incomplete data was highest in the age group 16-19 years and low before age 16 and after age 20. Such a distribution fits in with the above reasoning. The persons appointed to distribute and collect the forms had actually been asked to supply the name, age, etc. of those who had not menstruated yet. Though the series do include a small number of girls who had not yet menstruated, the number is so small that the large majority of teachers cannot have had occasion to inform themselves on this point. This must be kept in mind when the figures are considered.

Another source of error which must be taken into account is that replies will vary according to the age of the girls at the time they fill out the form. The mean age will not be high if young girls are asked. This error will probably disappear if older women are questioned, but then there is the risk that they will have forgotten the events of their youth and therefore will give incorrect replies. In order to illustrate the significance of age a table is appended which is based

Table 4. Number of women who respectively have and have not had menarche up to different chronological ages, and means for the age at menarche in a total of 9,416 women. Adapted from *Malmio*, 1919.

Age	Women who have had menarche			Women who have not had menarche	
	Number	% of total number	Mean age	Number	% of total number
13	286	3.0	12.3	9130	97.0
14	992	10.5	13.2	8424	89.5
15	2483	26.4	14.0	6933	73.6
16	4538	48.2	14.7	4878	51.8
17	6552	69.6	15.2	2864	30.4
18	7949	84.4	15.6	1467	15.6
19	8801	93.5	15.9	615	6.5
20	9134	97.0	16.0	282	3.0
21	9345	99.2	16.1	71	0.8
22	9388	99.7	16.2	28	0.3
23	9401	99.8	16.2	15	0.2
24	9410	99.9	16.2	6	0.1
25	9411	99.9	16.2	5	0.1
33	9416	100.0	16.2	0	0

on Malmio's data. He studied women who had born children and therefore must have menstruated. Table 4 gives the latest age at which information was given and the mean age at menarche of the women who had menstruation at that age, and the percentage of women who had not yet menstruated at that age. The table reveals the obvious fact that the mean age at menarche rises the older the women questioned. It is only after 21 years that the mean age at menarche no longer changes much. At that time as few as 0.8 per cent have not menstruated, and such a small number cannot affect subsequent figures. It may be said that the figures in table 2 might be inaccurate, but the main thing is that they illustrate a trend, viz. that the questions must be asked and the answers given when the women have acquired a reasonable maturity. If not, one must expect a large percentage who have not menstruated.

The girls studied in the present investigation were classified as follows:

- A. Those who attended the same school both before and after menarche.
- B. Those who attended different schools of the same type (i.e. coeducational or girls' school) before and after menarche.

C. Those who the year before menarche attended a school of a type different from the one they were attending when filling out the form.

Since, as shown above, the age at the time of questioning affects the mean age at menarche, the figures are consistently given in 1-year groups of the age when questioned.

For those who attended the same type of school before and after menarche (table 5) it will be seen that the mean age at menarche in

Table 5. Mean age at menarche for girls who the year preceding this event attended the same type of school as when information was given.

Age when questioned	Before and after menarche			
	same coeducational school	M	same girls' school	M
Number		Number		
13 - 14	—	—	6	(12.57)
14 - 15	—	—	63	13.26
15 - 16	14	(13.70)	454	13.51
16 - 17	143	13.54	549	13.68
17 - 18	208	13.71	519	13.72
18 - 19	154	13.81	155	14.09
19 - 20	62	14.27	9	(14.10)
20 - 21	9	(14.28)	2	(12.77)
21 - 22	1	(14.44)	—	—
Total	591	13.76	1757	13.67

the coeducational school series is somewhat higher than in the girls' school series. But this negligible difference may be due to random variation and can confidently be attributed to the fact that the girls in coeducational schools were slightly older than those in girls' schools when the information was supplied. Taking each age group separately, one finds that only in the 18-19 years group have the girls in coeducational schools a lower mean age at menarche than the girls in girls' schools. The difference amounts to 0.28 ± 0.12 and is consequently not statistically significant.

For those who attended different schools of the same type before and after menarche conditions are much the same (see table 6). In this case, however, the average age at menarche is a bit higher in the coeducational school series, but here the girls were also slightly older when questioned than those in the girls' school series. The differences between the ages at menarche in the 16-17 years group is significant

Table 6. Mean age at menarche for girls who the year preceding this event attended a different school of the same type as when information was given.

Age when questioned	Year preceding menarche			
	Other coeducational school		Other girls' school	
	Number	M	Number	M
14 - 15	1	(12.23)	2	(12.82)
15 - 16	7	(13.63)	21	12.60
16 - 17	41	13.69	33	12.67
17 - 18	95	13.78	39	13.19
18 - 19	85	13.87	16	13.53
19 - 20	51	14.19	6	(13.50)
20 - 21	18	13.93	—	—
21 - 22	3	(13.73)	—	—
Total	301	13.86	117	12.99

and amounts to 1.02 ± 0.24 , the girls' school series being the youngest. Pointing in the same direction, the difference in the 17-18 years group is 0.59 ± 0.20 years. Since only these groups display significant differences it would be unjustified to assert that girls from girls' schools begin to menstruate earlier than those from coeducational schools.

In group C (see table 7), lastly, the age at menarche is on the whole the same in the two series.

Table 7. Mean age at menarche for girls who the year preceding this event attended a school of a type different from that when information was given.

Age when questioned	Coeducational school the year before menarche - girls' school when questioned		Girls' school the year before menarche - coeducational school when questioned	
	Number	M	Number	M
14 - 15	1	(13.68)	3	(12.02)
15 - 16	17	13.10	14	(13.13)
16 - 17	37	13.35	61	13.51
17 - 18	30	13.79	99	13.56
18 - 19	28	13.99	97	13.60
19 - 20	8	(13.74)	76	13.89
20 - 21	2	(14.77)	20	14.16
21 - 22	—	—	5	(14.28)
22 - 23	—	—	1	(13.00)
Total	123	13.62	376	13.64

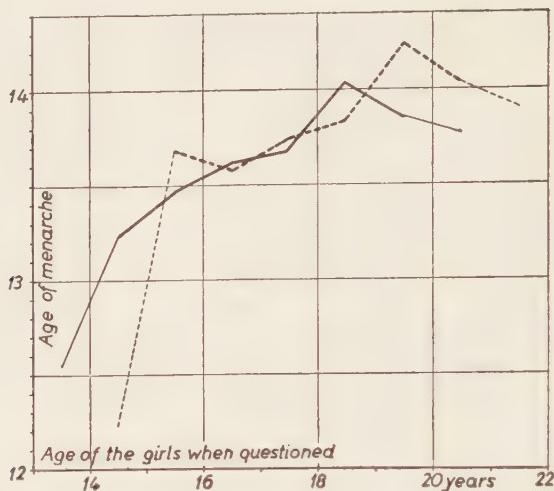


Fig. 1. Mean age at menarche distributed according to age when information was given for girls who attended coeducational schools (dotted line) and girls' schools (solid line). Those who went to the same school and those who changed schools are both included. The thin parts of the lines represent groups containing few subjects which may be inaccurate.

By combining groups A and B one gets the age distribution illustrated in figure 1. It shows that there is no very great difference between the groups, but that girls in coeducational and in girls' schools agree rather well with respect to the age when menstruation began.

With regard to the statement made above concerning data from the literature, it should be noted that a number of series are not representative and that the sources of error mentioned here have not been discussed. By and large, however, one gets the impression that menarche tends to come earlier in our times. But the data in the literature may be so fraught with errors that it would be unwise to draw definite conclusions in this respect. The statistical difficulty of obtaining comparable materials is probably greater than has generally been surmised. The possibility must therefore be taken into account that the difference which is mentioned in some works between bygone days and our times is imaginary and referable to errors of the type discussed above.

Salo's result has in any case not been confirmed. It is possible that in his case the coeducational school series and the girls' school

series had deviating distributions. As Salo has not published a full account of his material and methods the point cannot now be settled. One thing is certain, however, viz. that the difference which Salo claims to have found between Finnish girls from coeducational schools and from girls' schools with regard to the age at menarche does not apply to Swedish girls. It would therefore be pointless here to discuss to what extent the presence or absence of boys at school might affect the sexual development of schoolgirls. It should be noted, too, that girls in girls' schools also meet boys, and that the type of school would therefore be unlikely to have much effect on the development of girls. If there were such a difference it is theoretically rather improbable that it would affect the onset of menstruation.

Summary.

It has been claimed (*Salo [1947]*) that the menarche occurs earlier in girls attending coeducational schools than in those attending purely girls' schools. Girls from both types of schools were therefore investigated in this respect. A comparison of the two categories showed no significant difference. It is important, as shown, that the girls compared are of the same age when they answer the question of their age at the menarche, and that the negative answers too are registered.

Résumé.

On a prétendu (*Salo [1947]*) que la menarche arrive plus tôt chez des jeunes filles allant aux écoles mixtes que chez celles allant aux lycées de jeunes filles. Des jeunes filles de ces deux types d'école furent par conséquent examinées sous ce rapport. Une comparaison entre les deux catégories n'offrit pas de différence significante. Il est important, comme nous avons démontré, que les jeunes filles comparées soient du même âge quand elles répondent à la question de leur âge à la menarche et que les réponses négatives soient aussi enregistrées.

Zusammenfassung.

Es ist behauptet worden (*Salo [1947]*), daß die Menarche bei Mädchen, welche eine Gemeinschaftsschule besuchen, früher auftritt als bei solchen, welche eine ungemischte Mädchenschule besuchen. Mädchen der beiden Schultypen sind deshalb unter Bezugnahme

hierauf untersucht worden. Ein Vergleich der beiden Kategorien ergab keinen nennenswerten Unterschied. Wie gezeigt wird, ist es bedeutungsvoll, daß die miteinander verglichenen Mädchen dasselbe Alter haben, wenn sie die Frage nach ihrem Alter bei der Menarche beantworten, sowie daß die negativen Antworten ebenfalls registriert sind.

LITERATURE

- Backman, G.: Acta anat. 4, 421, 1947. – Bolk, L.: Akademie v. Wetenschappen 26, 650, 1923. – Breipohl, W.: Zentralbl. f. Gynäk. 61, 1335, 1937. – Van Castricum, M.: South African M. J. 20, 594, 1946. – Curjel, D.: Indian J. M. Res. 8, 366, 1920. – Dahlberg, G. and S. Akesson: Acta obst. gyn. Scandinav. 10, 63, 1930. – Engelman, C. J.: New York M. J. 75, 221, 1902. – Engle, E. J. and M. C. Shelesnyak: Human Biol. 6, 431, 1934. – Essen-Möller, E. Zentralbl. f. Gynäk. 30, 453, 1906. – Frommolt, G.: Rassenfragen in der Geburtshilfe. Leipzig 1936. – Heyn, A.: Ztschr. Geburtsh. u. Gynäk. 82, 136, 1920. – Gini, C. and Orchi, P.: Ricerche sulle famiglie numerose. Vol. 1, Rom 1939. – Kennedy, W.: J. Obst. a. Gynec. Brit. Emp. 40, 792, 1933. – Krieger, E.: Die Menstruation. Berlin 1869. – Lennér, A.: Acta obst. gyn. Scandinav. 24, 113, 1944. – Malmio, H. R.: Über das Alter der Menarche in Finnland. Helsingfors Diss. 1919. – Salo, A.: Suomalaisen kasvatuksen peruskysymykset. Del II. Helsingfors 1947. – Sanes, K. I.: Am. J. Obst. a. Gynec. 73, 93, 1916. – Samuelson, S.: Acta obst. gyn. Scandinav. 22, 33, 1942. – Schaeffer, K.: Arch. f. Gynäk. 84, 657, 1908. – Schlichting, X.: Arch. f. Gynäk. 16, 203, 1880. – Skerlj, B.: Arch. f. Gynäk. 159, 12, 1935. – Yamasaki, M.: Zentralbl. f. Gynäk. 33, 1291, 1909.*

THE MORTALITY RATE OF CANCER YESTERDAY AND TODAY

By GUNNAR DAHLBERG

It is well known with regard to malignant neoplasms that they may be secondary to irritation (cancer of the abdominal skin in Kashmirians; occupational cancer in aniline workers). In order to estimate the influence of environmental factors in such cases it is interesting to compare the incidence of malignant neoplasms in different populations. Such an analysis is presented in table 1. The table reveals that there exist some differences, but none that would warrant firm conclusions. The same thing appears in figures 1 a and b. Any differences that there are might very well be due to variations in the accuracy of records. It is anyway impossible to say if these differences are real or due to variation as to the accuracy of the records. The differences are most pronounced for men. The records for women seem to show better agreement. None the less

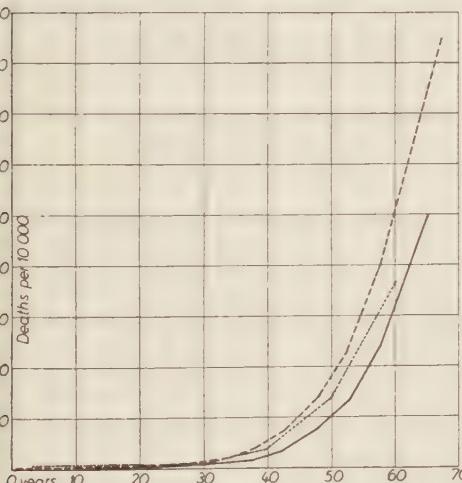


Fig. 1a.

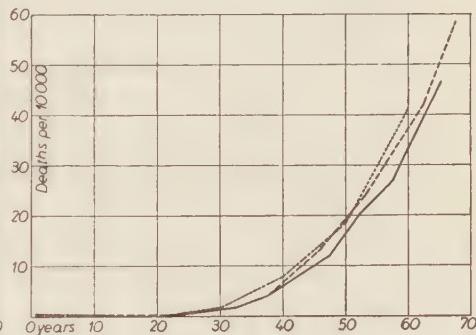


Fig. 1b.

Fig. 1a. Deaths from cancer and malignant tumours among men in Sweden 1945 (whole line), in England and Wales 1947 (broken line) and in Denmark 1948 (dotted line). *Fig. 1b.* Deaths from cancer and malignant tumours among women in Sweden 1945 (whole line), in England and Wales 1947 (broken line) and in Denmark 1948 (dotted line).

Table 1. Number of deaths from cancer and other malignant tumours per 10 000 inhabitants in different age groups and in different countries.

it seems impossible to draw any conclusions without extensive statistical analysis and knowledge of the conditions in different countries. We therefore pass on to an attempt to analyze the figures for Sweden.

Just over 10 years ago Professor Folke Henschen felt that the number of cancer cases at autopsy was increasing. Mainly for this reason Dr. Sven Hammarström consulted me and later studied the mortality rate of cancer at my institute, partly using Henschen's autopsy material, partly official statistics. Hammarström's thorough investigation, which was published in 1942, showed that the increase could not be at all significant. The increase found in the absolute number of deaths in cancer could be attributed to the greater longevity, one result of which was that there were more elderly people in this country. The higher birth rate formerly was of course a contributory reason for the larger number of elderly people. Hammarström's monograph contains a review of the early literature on this problem. It reveals that necropsy material is more satisfactory because it permits a more exact diagnosis. By studying such material it has been shown that some cases are not diagnosed before death. Necropsy material on the other hand suffers from the drawback that not all dead persons are autopsied. That is why it is difficult to use necropsy series for statistical analyses. For it is often impossible to estimate the size of the population from which they were drawn. Official statistics are not similarly affected and are therefore in some ways more reliable, even if some of the deaths listed under headings like decrepitude actually are missed cancer cases.

Hammarström's contribution to the literature of the 40's thus did not increase the anxiety for cancer mortality. To my mind the mortality rate of cancer has presumably declined, since ageing on the whole seems to take place more slowly and cancer in many ways is a disease of old age. It is common knowledge that the general mortality rate is much lower nowadays than it has been during preceding periods. It may be mentioned, for example, that King Gustavus I of Sweden died at the age of 64 years. At that time such an age was regarded as something quite unprecedented, all the more so as he was fit and capable of working to the very last. The lower death rate at high ages is probably an effect of slower ageing. As cancer to some extent is a disease of old age it may be surmised that its incidence will be lower in the more slowly ageing age groups. Strictly speaking it is of course impossible to prove that the lower death rate at high ages is a manifestation of slower ageing. For (apart

from the death rate) we have no means of measuring ageing. I have now re-examined the problem and, as I expected, it turns out that the mortality rate of cancer apparently has gone down somewhat.

Let me first say some words on the longer life expectancy for both the sexes together. It has increased and is now 74.5 years as against 63.5 years at the turn of the century, i. e. a gain of 11 years. This increase is as we know predominantly due to the sharp decline of the infant mortality rate that has taken place. It will be remembered that the probable expectancy of life is based on the median whereas the average term of life is based on the mean. Comparing now the mean term of life remaining at 50 years, we find that in 1901–1910 it was 23.2 years for men as against 25.0 years in 1941–1945, the difference being only 1.8 years. The corresponding figure for women was 1.5 years. Obviously this change in mortality and ageing for 50-year-olds cannot have a very great effect, but it is none the less justified to study the mortality rate of cancer. It must be kept in mind, of course, that our diagnostic facilities have improved greatly, particularly through roentgenography and the like, and probably have entailed a far greater augmentation of the number of recorded deaths in cancer than the decrease due to our moderate therapeutic advances. In view of this source of error one would rather expect that the mortality rate of cancer had risen. The annual number of deaths in cancer for the period 1921–1925 is given officially as 6,090 and for 1946 as 9,258. The picture takes on a somewhat different appearance, however, if we compare the percentages of cancer

*Table 2. The mortality from cancer among women in 1911 and in 1947.
The whole country.*

Age, years	1911		1947	
	Number of deaths	Per 10,000 of the population	Number of deaths	Per 10,000 of the population
0 – 5	—	—	1	0.03
5 – 10	1	0.03	1	0.04
10 – 15	1	0.04	—	—
15 – 20	3	0.1	2	0.1
20 – 30	12	0.3	26	0.5
30 – 40	99	2.8	162	3.0
40 – 50	289	9.7	512	10.5
50 – 60	584	22.4	900	22.8
60 – 70	856	43.2	1282	43.2
70 – w	1006	59.6	1979	86.7

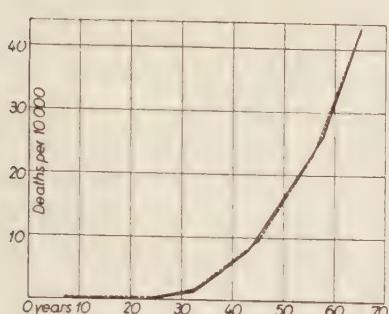


Fig. 2

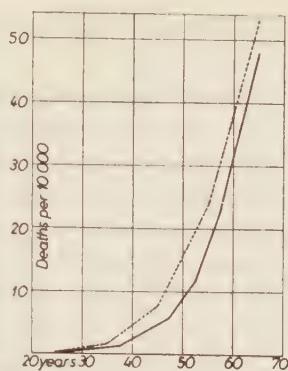


Fig. 3

Fig. 2. Deaths from cancer per 10 000 women of different ages in the year 1911 (dotted line), respectively in 1947 (whole line). Fig. 3. Deaths from cancer per 10 000 men of different ages in the year 1911 (dotted line) respectively in 1947 (whole line). The whole country.

deaths at different age levels. Table 2 and figure 2 give the number of deaths in various age groups per 10,000 for women in the whole population in 1911 and in 1947. It will be seen that there are no differences worth discussing. With regard to men we have table 3 and figure 3 which reveal a distinct decline. It should be noted that the diagrams give no death rates for cancer over 70 years of age. The reason is that

Table 3. The mortality from cancer among men in 1911 and in 1947.
The whole country.

Age, years	1911		1947	
	Number of deaths	Per 10,000 of the population	Number of deaths	Per 10,000 of the population
0 - 5	—	—	—	—
5 - 10	—	—	—	—
10 - 15	—	—	1	0.05
15 - 20	—	—	3	0.1
20 - 30	14	0.3	15	0.3
30 - 40	59	1.7	65	1.2
40 - 50	209	7.8	236	4.9
50 - 60	542	23.9	657	17.8
60 - 70	878	52.9	1302	48.1
70 - w	917	70.5	2087	108.3

in this age group the figures tend to be rather unreliable, because the frequency of deaths from decrepitude has gone down progressively as more exact causes of death have been substituted. With the χ^2 -test the difference in cancer mortality is significant for men ($P < 0.001$) but not for women ($P < 0.7$). The figures for cancer mortality given in the tables apply to the whole population. If we limit the comparison to the urban population, where the information of the causes of death is more reliable, the same differences are found. This being so we shall now consider the mortality rate for cancer of the breast. Since 1921 the appropriate figures have been broken out and given separately in official statistics, and the group is rather homogeneous. As has been brought out particularly well in an investigation by *Dorn* [1943], and by other authors too, cancer of the breast strikes single more often than married persons. Obviously this has nothing to do with any psychosomatic effect of the wedding; probably it is due to the simple fact that married women have far more pregnancies than single. For additional information the reader is referred to *Westberg's* investigation [1946] in which the problem is discussed at length. If we now compare the mortality rate of cancer of the breast in women in 1921 with that in 1945, we find that cancer of the breast had a higher incidence in the latter year. It would seem that the risk of cancer of the breast has been increasing of late, which in a way is natural considering that during the preceding decade women consistently had fewer pregnancies than formerly used to be the case. This higher risk of breast cancer is probably responsible for the fact that the mortality rate of cancer in women has not gone down despite the slower ageing. That is in any case the most natural explanation I can think of (table 4). As cancer of the breast is among the forms of cancer which are most accessible to treatment, the figures show that treatment has no very great effect on the death rate. In any case the increase of the death rate due to other factors seems to be greater than the decrease expected from the treatment.

For the sake of completeness we shall also consider the mortality rate of sarcoma. Prior to 1921, however, no data were published so we cannot go beyond that year in our analysis. Table 5 and figure 4 apply to women and display the difference in some high age groups. Table 5 and figure 5 for men also show a small difference. The difference is not significant by the χ^2 -test but illustrates the trend expected. (For men $P < 0.5$ and for women $P < 0.3$.)

Table 4. The mortality from cancer of the breast among women in the urban population in 1921 and in 1945.

Age, years	1921		1945	
	Number of deaths	Per 10,000 of the population	Number of deaths	Per 10,000 of the population
30 - 40	7	0.5	20	0.8
40 - 50	35	3.1	69	3.2
50 - 60	40	4.6	92	5.5
60 - 70	28	4.3	105	8.8
70 - w	48	10.5	90	11.0

In summing up it may be pointed out that the mortality rate of cancer has gone down for men but hardly for women. The mortality rate of sarcoma seems on the other hand to have decreased for both men and women. The position may perhaps be easier to grasp if it is noted that in 1947 the number of deaths in cancer among 50-60 years old men was 227 fewer than would have been the case had the 1911 mortality rate of cancer been applicable in 1947. The difference for all ages would be 209 men. For women in the 50-60 years age group the corresponding difference is only 15 deaths. For all

Table 5. The mortality from sarcoma among the urban population (both sexes) in 1921 and in 1945.

Age, years	Men				Women			
	1921		1945		1921		1945	
	Number of deaths	Per 10,000 of the population	Number of deaths	Per 10,000 of the population	Number of deaths	Per 10,000 of the population	Number of deaths	Per 10,000 of the population
0 - 5	1	0.1	5	0.4	1	0.1	1	0.1
5 - 10	—	—	4	0.5	1	0.1	1	0.1
10 - 15	—	—	—	—	1	0.1	—	—
15 - 20	1	0.1	1	0.1	1	0.1	3	0.3
20 - 30	8	0.5	7	0.3	5	0.3	2	0.1
30 - 40	3	0.2	4	0.2	5	0.3	11	0.4
40 - 50	3	0.3	10	0.5	8	0.7	8	0.4
50 - 60	4	0.6	6	0.4	14	1.6	13	0.8
60 - 70	12	2.8	18	1.9	12	1.9	25	2.1
70 - w	9	3.6	12	2.2	12	2.6	16	2.0

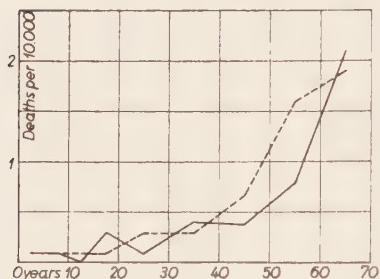


Fig. 4

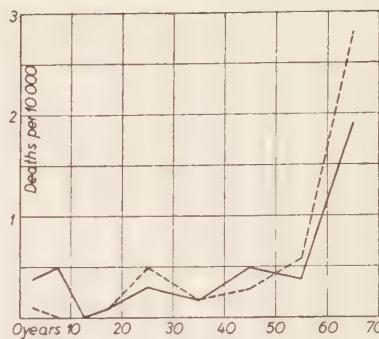


Fig. 5

Fig. 4. Deaths from sarcoma per 10 000 women of different ages in the year 1921 (broken line) and in 1945 (whole line). *Fig. 5.* Deaths from sarcoma per 10 000 men of different ages in the year 1921 (broken line) and in 1945 (whole line). (The urban population in both figures.)

ages the difference amounts to 695 more dead women than expected, but the latter figure includes the unreliable group above 70 years of age.

For sarcoma the theoretical number of deaths in 1945 is 80 men and the true figure 67. This means that there was a gain of 13 who survived. For women the corresponding figures are 106 and 80, i. e. a gain of 26 surviving women. Relative to past times we have consequently made a consistent gain owing to a decline in the mortality of cancer and sarcoma. It may be surmised that the gain will be more marked in the future. But the decrease cannot continue indefinitely, for ageing cannot diminish indefinitely.

Summary.

The diminishing death rate shown for the Swedish population does not seem to be due to a better treatment of malignant tumours but due to the increasing length of life, among other things possibly because of the breaking up of isolates. The problem is tentatively discussed in this paper.

Résumé.

La mortalité diminuée de cancer dans la population suédoise ne semble pas dépendre d'un traitement meilleur des tumeurs malignes mais de la durée de vie augmentée dépendant surtout de la rupture des limites des isolats. Dans ce travail l'auteur fait un essai de discuter le problème.

Zusammenfassung.

Die sich verringерnde Sterblichkeitsquote, aufgezeigt für die schwedische Bevölkerung, scheint nicht einer besseren Behandlung von malignen Tumoren, sondern der zunehmenden Lebensdauer infolge des Brechens der Grenzen der Isolate teilweise zuzuschreiben zu sein. Das Problem ist in dieser Arbeit versuchsweise besprochen worden.

LITERATURE

Dorn, Harold : Human Biol. 15, 73-79, 1943. – Hammarström, Sven : Acta med. Scandinav. 112, 568-584, 1942. – Westberg, S. V.: Prognosis of breast cancer for pregnant and nursing women. Håkan Ohlssons Boktryckeri, Lund 1946.

A NOTE ON THE DILEMMA OF HUMAN GENETICS

By GUNNAR DAHLBERG

On the opening page of his book "Sceptical Essays" Lord Bertrand Russell formulated what he feared would be considered a "paradoxical and subversive" doctrine: that one should not believe things which are not supported by a trace of proof. "I must of course admit", on went Lord Bertrand, "that if such an opinion became common, it would completely transform our social life and our political system. I am also aware that it would tend to diminish the incomes of clairvoyants, bookmakers, bishops, etc."

It has at any rate been claimed that this doctrine is essential to science; voices have even been heard to the effect that it should be raised to the status of a Divine Commandment—the Eleventh. The scientist, however, considers nothing proved if the facts allow a different interpretation.

When Mendelism first was widely recognized at the turn of the century attempts were made to copy animal experiments by drawing up pedigrees in which given characters or diseases occurred as the Mendelian laws would lead one to expect. Gradually it became clear that such pedigrees in many instances were selected on account of

a random accumulation of such character-bearers. Hence they were not very good evidence. One actually did not start solely from persons with the required characters and then studied their descendants; one knew that the character was present in the descendants of the persons one started from. Rather than a person the origin of the material was thus an entire family, and obviously then the number of character-bearers would be too high. In other words one put the cart before the horse and started with what was to be proved. Such pedigrees have nevertheless produced some results, particularly with regard to very rare characters.

We shall illustrate this criticism with a pedigree of mothers who had given birth to twins which is taken from a paper by Bonnevie and Sverdrup [1936]. Undoubtedly we are in this case dealing with a random accumulation of twin births. (That is also revealed by the

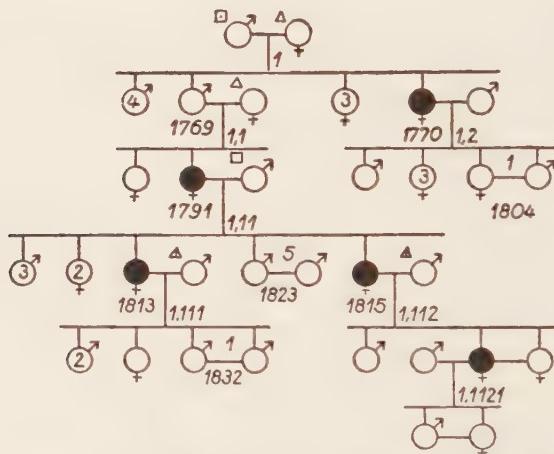


Fig. 1. Fragment of a pedigree after Bonnevie and Sverdrup [1926].

large pedigree of twin births of which the present one is part.) Moreover, as the authors pointed out themselves, the character of bearing twins is obviously not due to a sex-linked dominant gene.

Owing to criticism of this sort statistically informed persons have, when it is a matter of fairly common traits, long taken a negative attitude to pedigrees of the aforementioned type. They have instead begun to start from character-bearers that are unselected as regards

the properties of the family and then to assess the frequency of the trait in relatives. The latter procedure has for example been applied to mental disorders and epilepsy, but the results have not always been easy to interpret. Attempts have been made in such cases to introduce a varying penetrance or a more involved mode of transmission. But studies of this kind are based on doubtful concepts. It is assumed that the possessors of the character have the same genes and, therefore, produce offspring according to the same mechanism, which by no means is certain. Examples of such studies are those of Rüdin [1916] and Kallmann [1938] on certain mental diseases and Alström's [1950] on epilepsy.

However, there exist several examples which demonstrate that the same traits are inherited in different ways in different families (cf. Forssman's paper on Diabetes Insipidus [1945]) and actually caused by different genes. Forssman's paper contains 3 pedigrees in which the trait was sex-linked, while in the others it was transmitted by an autosomal dominant gene. The author considered this later gene to have a variable penetrance. Clearly the trait may also appear as a result of environmental influences. Every textbook on Human Genetics gives many examples of the same character being inherited in a different manner in different families and consequently produced by at least slightly different genes.

It seems justified to assume that a genetic disease so long as it does not become too rare arises through a series of mutations. It is likely, however, that the mutations have a slightly different character and give rise to different modes of transmission. It is further likely that the diseases occur in slightly different forms. However, even if they are inherited in different ways, the different mutations may produce practically identical signs and symptoms. Any variations in the signs and symptoms may perhaps only be brought to light through very penetrating clinical observation and merely manifest themselves in the intensity of the disease.

It should be remembered, moreover, that the same cause may produce disease manifestations of a slightly varying nature. An oft-cited example of this is that alcohol intoxication is attended by somewhat varying symptoms of drunkenness, although the cause is the same. One inebriate may be euphoric, another rather aggressive, while a third even may display signs of depression with paranoic ideas, etc. It is therefore unsafe to rely on the clinical examination exclusively. A gene may have varying effects in different genotypes.

Returning now to Lord *Bertrand's* maxim, we find that human genetics is in a dilemma, at least over the heredity of common traits. Ordinary pedigrees have the disadvantage of often giving rise to overrepresentation of character-bearers and hence of exaggerating the importance of heredity. Statistical analyses based on unselected character-bearers, on the other hand, presuppose that all the traits considered are inherited in the same way, which by no means is certain and in some cases completely wrong. Both methods must therefore be used circumspectly. At present there exists no completely satisfying solution to the problem.

This holds good from a theoretical point of view which I suggested in my paper "Genetics of convergent strabismus" [1951]. If one only wishes to know the average risk for a disease to appear among the children the method of starting from not chosen character-bearers of course is all right.

LITERATURE

Alström, Carl Henry: A study of epilepsy in its clinical, social and genetic aspects. Ejnar Munksgaard, Copenhagen, 1950. — *Bonnevie, Kristine and Aslaug Sverdrup:* J. Genetics 16, 125–188, 1926. — *Dahlberg, Gunnar and W. Nordlöw:* Acta Genet. and Statist. Med. 2, 1–14, 1951. — *Forssman, Hans:* On hereditary diabetes insipidus. Håkan Ohlsson, Lund 1945. — *Kallmann, Franz J.:* The genetics of schizophrenia. J. J. Augustin, New York 1938. — *Rüdin, E.:* Zur Vererbung und Neuentstehung der Dementia praecox. Springer, Berlin 1916.

From the State Institute of Human Genetics and Race Biology
(Head: Professor Gunnar Dahlberg),
Uppsala, Sweden

THE SOCIAL OUTLOOK FOR CHILDREN OF DIVORCEES

By EDITH OTTERSTRÖM, M. D.

In most countries, even the U.S.A., it is possible to get a divorce to prevent the holy wedlock from degenerating into a holy deadlock. The divorce rate of course varies greatly, depending as it does on a variety of laws and customs and, not least, religious taboos. Moreover the available statistics on divorce are not comparable.

The childrens' fate is one of the factors to be considered in discussions on divorce. A number of authors (*Braun, Kistler, Madörin, d'Okolski, Pflugk, Rickhard, Rothschild, Strobel*) have taken up the legal and social aspects of the problem, but they have merely given the percentages of divorcee's children against whom action has been taken by the authorities.

Haffter [1948] seems to be alone in having followed up the later development of a series of divorcee's children—103 boys and 107 girls. He saw things mostly from a psychiatrist's point of view and attempted by means of personal interviews to form an opinion regarding the mentality of the children and the extent to which it had been influenced by their parents' divorce.

After due consideration he concluded that his investigation on the whole tended to disprove or invalidate the generally held view that a parental divorce always is harmful to the children. He maintained that one third of the cases of unfavourable development were due to an inherent abnormality in the children themselves, such as psychopathy, backwardness and epilepsy. He thought that the age of the children at the time of the divorce was especially important; schoolchildren, particularly during puberty, were most exposed to risks.

Thus, so far as I can find, there exists no objectively founded treatise on the social outlook for a considerable number of divorcee's children.

It is desirable in investigations such as these to operate with well-defined characters and compare them with the normal populations', i.e. to study matters that lend themselves to objective recording, e.g. detention for drunkenness, conviction for crimes, etc. Psychiatric investigations, on the other hand, have the drawback that they tend to become biased and therefore are unconvincing.

Materials.

The present investigation is based on a series of 606 families in which man and wife were legally divorced at Malmö¹⁾ in the period 1920–1931 and the existence of children is mentioned in the Divorce Courts' records. Table 1 gives the total number of divorces granted during that period and the number of divorced families in which there were children. It appears that one third of the dissolved marriages were childless.

The figures for the number of children per family could in some cases be supplemented from the records of Malmö Town Council

¹⁾ The third largest town in Sweden.

Table 1. Total number of divorces in Malmö during the period 1920–1931.

Year	All dissolved marriages (divorces) during different years	Dissolved marriages with children	
		Number of marriages	Number of children
1920	64	45	94
1921	59	47	126
1922	77	54	183
1923	75	47	120
1924	84	60	154
1925	69	46	96
1926	72	47	118
1927	79	53	140
1928	82	51	113
1929	85	57	109
1930	68	42	99
1931	89	57	112
Total	903	606	1464

regarding beneficiaries of social welfare¹⁾). But these did not always give full names or dates of birth, and the children concerned had then to be excluded from the investigation. Quite possibly the exact number of children was not secured for all the families. It is very likely that some were grown up and had left home at the time

Table 2. Distribution of the number of children on different families at the separation or the divorce of the parents.

Number of children per family	Number of families	Total number of children
1	240	240
2	144	288
3	97	291
4	59	236
5	28	140
6	15	90
7	13	91
8	5	40
9	2	18
10	3	30
Total	606	1464

¹⁾ All Swedish burroughs keep record of people who have received aid from public funds.

of the separation or, even if young, were domiciled elsewhere, e.g. with grandparents or a guardian.

Ultimately the total number of children in the families numbered 1464. At the time of separation 240 families, 39.6 per cent, had 1 child (table 2). At most 4 children occurred in 540 families and more than that were present in 66 families. Of these 10 had as many children as 8, 9 or 10. The average number of children per family was 2.4. It is of interest to note the age of the children at the time of their parents' separation or divorce. As appears in table 3 most

Table 3. Age of the children at the time of their parents' separation or divorce.

Age in years	Boys	Girls
0-1	48	37
1-2	32	42
2-3	40	37
3-4	38	29
4-5	36	40
5-6	33	43
6-7	31	37
7-8	38	31
8-9	32	26
9-10	33	37
10-11	27	36
11-12	23	38
12-13	29	23
13-14	27	19
14-15	36	33
15-16	37	28
16-17	30	19
17-18	21	20
18-19	23	20
19-20	19	22
20-21	15	10
21-22	6	9
22-23	7	5
23-24	5	2
24-25	2	7
25-26	4	3
26-27	2	2
27-28	2	1
28-29	—	1
Total	676	657

of the children were then less than 15 years old (503 boys and 508 girls). More than half of these were too young for school, i.e. they were below 7. Of age were 28 youths and 30 girls.

The Divorce Court's records in one third of the cases stated that the decree was granted on the grounds of incompatibility, but it is difficult to tell what in the individual case is hiding behind this official term.

To be sure, the true reasons for the divorce were occasionally given, e.g. the husband's abuse of alcohol in 59 cases (10 per cent)—a figure which probably is far too low. Bigamy, adultery or transmission of venereal disease to the spouse were held responsible in 51 cases, the first reason being mentioned once only. Other grounds for divorce were criminality in 10 cases and mental disease in 16 (12 wives and 4 husbands). Many times more than one reason was given, such as abuse of alcohol combined with financial difficulties, the husband's inability to maintain his family, his niggardliness with housekeeping money, the wife's lack of thrift, a bad temper on the part of either or both spouses, etc. In 4 cases the marriage was arranged to legalize an expected child; the parents never cohabited after the wedding and applied for a divorce soon after the child was born. No reason for the divorce is given in respect of 20 families.

In order to get some idea of the childrens' social status in the parental home all the families in the series, 606, were classified in social groups according to the father's occupation, as specified in the Divorce Court's records. The subdivision was made in conformity with the norms adopted in the official election statistics for 1921–1940 with respect to Malmö, Hälsingborg, Lund and Landskrona townships which together make up one election district. It appears that social group I (prosperous people, upper middle class) is clearly underrepresented (4.8 per cent), social group II (lower middle class, skilled labourers) is more or less in parity (29.4 per cent) and social group III (unskilled labourers, vagrants) is rather overrepresented (65.8 per cent). The corresponding normal proportions for these towns are 9.3, 28.2 and 62.5 per cent.

Another key to the environment is provided by information concerning the social aid paid out to the family. A check-up in Malmö's social records revealed that 55.8 per cent of the families had received some form of aid: 17.8 per cent of these had already such aid before the divorce, and not quite half the families (44.2 per cent) had not found it necessary to apply for help. Not being

domiciled in Malmö, 9 families were not looked up in the social welfare records. It is evident, thus, that more than half of these families were in economic difficulties and the husband had been a poor provider.

In addition the parents were sought in the penal register¹⁾ and the men in the records of the local temperance board at Malmö. It turned out that 19.8 per cent of 606 fathers had been convicted of a felony. Compared with *Dahlberg's* [1943] figures for the male population aged 55–60 years of Malmö (9.9 per cent) this figure is twice as high. Compared also with *Dahlberg's* figure for divorced males of the same age among the Swedish urban population, 17.5 per cent, which figure exceeds the mean for men in towns, our figure is still very high. Among the mothers 1.8 per cent had been convicted. *Dahlberg's* figure for 50 years old women in towns, 1.1 per cent, is far lower. (Being expatriated at the time 7 women were not looked for in the various records.)

The fathers but not the mothers were sought in the records of the local temperance board at Malmö. Regard was also paid to notations concerning abuse of alcohol in the Divorce Court's records in cases when the woman had applied for divorce wholly or partly on such grounds. Whenever the same information was entered in two or more sets of records only one entry was counted. It turned out that 96 men (15.8 per cent) were addicted to alcohol. This figure is a bit excessive compared to *Dahlberg's* [1942] risk figures for men from Malmö during the years 1929–1933 and 1934–1938 of, respectively, 10.0 and 12.0 per cent at an age of 55–60 years.

We may evidently conclude that two thirds of this series of families came from social group III, more than half of them were very poor, and the men had records of criminality and intemperance that documented their inferiority to the male population at large in Malmö. As regards criminality the same applied to the women.

Prognosis.

A study was made of the prognosis for the social status of children when grown up; it was based on information supplied by the authorities concerned. Every person included in the prognostic

¹⁾ In Sweden a central penal register is kept of persons who are sentenced to penal servitude or imprisonment or, for petty larceny, to payment of fines; and of persons given probationary sentences or found guilty of a crime or felony but, on grounds of insanity, not convicted thereof; and of persons sentenced to forced labour.

study was followed up from the time of the parental divorce to January 1st, 1949; if they died or left Sweden before then, to the date in question. The factors considered were *a) measures taken by the Child Welfare Board, b) criminality* (entered in the penal register), *c) abuse of alcohol* (subjected to measures from the Temperance Board or sentenced for drunkenness) and *d) receipt of social aid.*

For the statistical analysis was used Dahlberg's method of calculating annual and cumulative risks, whereby the results would be rendered comparable with his for the total population.

All the children in the basic series could obviously not be followed up. To begin with all those children were excluded who could not be traced as well as all those whose full names and dates of birth were not available. What then remained was 1333 persons, 676 males and 657 females from 585 families.

Measures by the Child Welfare Board.

The first phase of the prognostic analysis was to see if the person concerned had incurred measures by the Child Welfare Board. The only measures considered were "warnings" and "detention for schooling at a reform school". Measures like "public care", etc. which are unrelated to delinquency were not taken into account.

Only those boys and girls who remained in Malmö during their adolescence or removed to one of the larger towns were followed up. Most of the others at an early age left with the mother for her home province. As it is very likely that the rural Child Welfare Boards do not function as efficiently as those in the towns—for lack of trained officers—and also keep less reliable records, it was considered unsuitable to include children of the latter category about whom only incomplete information would be obtainable in any case.

The number of persons sought in the records of the Child Welfare Board therefore numbered 1253, 634 boys and 619 girls. Among the boys 9.6 per cent had been the object of the most lenient form of measure, a warning, while 6.6 per cent had been detained for reform schooling and separated from their homes. There are no statistics available which give the frequency of warnings. However, in my previous investigation into the prognosis for children that because of delinquency had been sent to reform school by the Child Welfare Board of Malmö town (published 1946) I found a frequency of 1.5 per cent for boys and 0.97 for girls. Hence, compared to the former of these figures, our frequency for boys from divorced families, 6.6 per

cent, is at least 4 times as high as the risk for Malmö boys in general of being sent to reform school.

Among the girls 1.1 per cent had received a warning while 2.1 per cent had been sent to reform school. In comparison to the above figures cited from my previous investigation, it would seem that girls from divorced families run twice as large a risk of being taken care of for reform schooling as other Malmö girls.

It should be added that in several cases, about one third, the Child Welfare Board's action, particularly the warning, was administered before the parents were divorced. Owing to the small number of cases it would be impractical to classify them according to whether action was taken before or after the divorce; and, besides, the value of such a classification is very doubtful. Yet it is probable that the environment already was unsuitable and the children badly taken care of before the divorce.

Criminality.

Men. Considering first the annual risk for men at different ages, we find that it was highest for the 15–18 year-olds (1.8 per cent, just as in the population at large even if the figure for the same age

Table 4. Annual risk and calculated cumulative risk in per cent of committing a crime for men and women with divorced parents.

Age, years	Men	Women
Annual risk, %		
15–18	1.82	0.20
18–21	1.78	0.26
21–25	0.91	0.08
25–30	0.48	0.11
30–35	0.35	0.05
35–40	0.47	—
40–45	0.39	—
45–50	0.61	—
Calculated cumulative risk, %		
18	5.4	0.6
21	10.3	1.4
25	13.5	1.7
30	15.6	2.3
35	17.1	2.5
40	19.0	2.5
45	20.6	2.5
50	22.9	2.5

group of the latter is much smaller, viz. 1.3 per cent according to *Dahlberg* [1943] (see table 4). Later on the annual risk for men with divorced parents declines, although it remains quite high relatively speaking. The same applies to the cumulative risk which at 50 years is 22.9 per cent. In other words: More than one fifth of the men with divorced parents will be convicted of a felony if they live till they are 50 years old. Compared with *Dahlberg's* aforementioned risks for the male population of Malmö at the same age, 9.9 per cent, it appears that boys from divorced families when they are grown up run twice the risk of others of getting into the penal register.

Women. The risk for women in general of committing a crime is, as we know, very small. The annual risks for 18–21 year-olds, which is the highest, is 0.07 per cent (*Dahlberg* [1943]). The cumulative risk at 50 years is 1.1 per cent for the total female population. This means that just over 1 woman in a hundred will have been convicted of a crime by the time she is 50 years old. On the other hand, the annual risk for women with divorced parents is thrice as high as the normal, and the cumulative risk at 50 years, being 2.5 per cent, is at least twice the normal figure.

Alcoholism.

To ascertain whether the persons concerned had at any time been addicted to alcohol, the temperance boards in the towns where they had lived as grown-ups were asked about them, and the Control Board at Stockholm was queried whether they had at any time been fined for drunkenness. The former authorities were only asked about men born 1900 or later since temperance boards were only set up in 1916. The Control Board at Stockholm did not start keeping central records until 1932, so they were only queried about those born 1917 or later. No attempts were made to ascertain the incidence of alcoholism in the females in our series, since the risk for women of becoming addicted to alcohol is so small that a statistical analysis would have been of very little value with such a small number of cases.

For the men in this series the annual risk of being subjected to intervention from the temperance board was highest in the 20–35 years age group, being almost 1 per cent for both 20 and 25 year-olds (see table 5). In relation to *Dahlberg's* [1942] figure for the male population of Malmö, which at age 35 is 0.27 per cent, the annual risk in our series was almost 4 times as great. The cumulative risk was also high; being 17.0 per cent at 40 years it was almost 3 times the

Table 5. Annual risk and calculated cumulative risk in per cent of being subjected to intervention from the Temperance Board or sentenced for drunkenness for men born in 1900, respectively in 1917, whose parents were divorced.

Age, years	Men	
	Reported for alcoholism	Sentenced for drunkenness
Annual risk, %		
15-20	0.46	0.73
20-25	0.99	1.37
25-30	0.74	0.97
30-35	0.99	—
35-40	0.53	—
40-45	0.50	—
Calculated cumulative risk, %		
20	2.3	3.6
25	7.0	10.0
30	10.4	14.3
35	14.8	14.3
40	17.0	—
45	19.1	—

mean frequency (6.1 per cent) for the urban population of the same age in the years 1934-1938 (*Dahlberg* [1942]).

For these men the annual risk of being sentenced for drunkenness reached a peak in the 20-25 years group (1.37 per cent) and at 35 years 14.3 per cent had had this experience. As a comparison it may be mentioned that the cumulative risk at the same age for the male urban population was 7.3 per cent in 1938 and 10.9 per cent in 1942 (*Dahlberg* [1944]).

Vagrancy.

Steps were actually taken to ascertain whether the subjects had been warned for vagrancy, but only a small number—3 women and 10 men—had incurred that risk, and a statistical analysis based on so meagre data would produce no significant results.

Social Aid.

We now come to the problem as to what extent children from divorced families are able to fend for themselves when grown up and to what extent they must rely on assistance from the community (see table 6). Ordinarily the need for social aid rises with the years. Apparently this was not the case with the children in our series for

Table 6. Annual risk and calculated cumulative risk in per cent of needing social aid for men and women with divorced parents.

Age, years	Men	Women
Annual risk, %		
15-20	1.50	1.86
20-25	2.43	2.35
25-30	2.49	1.29
30-35	1.42	1.11
35-40	1.69	1.05
40-45	0.71	0.51
45-50	—	—
Calculated cumulative risk, %		
20	7.3	9.0
25	18.0	19.2
30	27.7	24.3
35	32.7	28.4
40	38.2	32.0
45	40.4	33.8
50	40.4	33.8

whom the need was greatest in the 20-30 years group of men and in the 20-25 years group of women. This discrepancy from the total population might be due to need for economical assistance for studies. For it is a fact that the economical resources of a family deteriorate after a divorce and the children have to do without vocational training that they otherwise would have received, so that society must intervene. This factor—the lack of vocational training—may also be the reason why as many as 40 per cent of the men and 34 per cent of the women had already been assisted in some form by the time they were 45 years old. Corresponding normal figures do not exist and it should be added that these percentages are maximal because no classification was done, and temporary assistance and unemployment compensation was included.

It seems that the women from divorced families perhaps did a little better than the men, but that does not mean so much when it comes to judging their personal qualifications: most of them were married and economically more or less dependent on their husbands.

Social Status.

Lastly we shall briefly consider the social standing in adulthood of these children from divorced families (see table 7). As regards the

Table 7. The social standing of the sons compared with that of their fathers at the divorce.

Fathers / Sons	Social classes		
	I + II	III	Total
I+II	127	75	202
III	78	340	418
Total	205	415	620

men the table reveals on the whole the same distribution in classes as the general population. One third (202 of 620) belonged to social classes I and II while two thirds (418 of 620) came from social class III. (There being no information as to their employment or trade 56 men could not be included in this analysis.) Comparing the eventual social position of the sons with that of their fathers at the time of the divorce, we find that not quite two thirds (127 of 202) of those whose fathers belonged to class I and II retained that position while 75 of 202, i.e. more than one third, came down ($37.1 \pm 3.4\%$). Among those men whose fathers came from social class III not quite one fifth ($18.7 \pm 1.9\%$) had improved their position. It appears, therefore, that more than one third of all the sons had come down in the world and that one fifth only had managed to work themselves up from the class they were born into. The difference, 18.4 ± 3.9 , is statistically significant.

As mentioned in the introduction Haffter [1948] demonstrated (though his series was not particularly numerous) that his children of divorcee's had approximately the same fate. This might be attributable to the economic straits which families often get into after a divorce and from which the children primarily will suffer.

The social status of the women was not analyzed statistically. Their professions, if they had any, when unmarried could not be ascertained. Most of them were married (503 of 639 with known civil status) and, taking their husbands' social status and conditions, their position in society could not be accepted as a measure of their capability. Among these 503 as many as 36, i.e. a bit over 7 per cent, were divorced. Among the 612 men whose civil status was known 472 had married and 41 of these were divorced, i.e. just under 10 per cent. Evidently the divorce rate was somewhat higher for the men than for the women (but not particularly high for either sex).

Age.

After the preceding general review a few questions arise. First, does the age of the children at the time of the parental divorce influence the children's later development? It seems likely that the earlier the parents have their divorce, and thus the younger the children, the less would they be capable of understanding either the conflict between the parents or the subsequent change of environment. The opposite may be said of the children who were over school age or even grown up at the time and who throughout their childhood and adolescence had suffered from the oppressive home atmosphere and perhaps even had participated in their parents' recriminations. Have the older children led more twisted and unsuccessful lives than the young group and more than the intermediate group?

It should be remembered, however, that even if a marriage has broken after many years it does not mean that all the years of married life have been a single conflict. No doubt many years may have been most harmonious and beneficial to the children until, suddenly, for one reason or another, something happened that shook the very foundations and made the ties break. The father or the mother might have fallen in love with someone else, the family might have fallen upon evil times economically, either party might have committed a crime. On the other hand it is often claimed that experiences in early childhood play a significant role in the development of the child and of its future character. This is far from proved, but the possibility must be taken into account. Consequently there is some point in studying the age of the children at the time of the parents' divorce. For this purpose we classified the children in 4 groups according to their age at the time of their parents' separation or divorce, viz. younger than 4 years, between 4 and 9 years, between 9 and 15 years, and 15 years and older.

Measures taken by child welfare boards in relation to the age classification must be omitted here. Behavioural abnormalities before age 7 are not regarded as delinquency and therefore call for no intervention by the authorities; and, as long as the old Child Welfare Act was in force, nobody over 15 could become the object of an intervention. As pointed out elsewhere the series was not sufficiently numerous to permit further subdivision than that.

Male Criminality was, as table 8 shows, characterized by higher risk figures the younger the children were at the time of their parents' divorce. This was true of both the annual and the cumulative risk

Table 8. Annual risk and calculated cumulative risk in per cent of committing crimes for men and women divided into groups according to their age at the divorce of the parents.

Age, years	Age of the children at the divorce of the parents, years			
	Under 4	4–9	9–15	15 and over
Men				
15–18	2.42	2.85	1.35	0.78
18–21	2.15	1.99	1.44	1.59
21–25	0.69	0.91	0.82	1.13
25–30	0.58	0.96	0.42	0.14
30–35	0.80	0.35	0.17	0.42
35–40	—	—	0.61	0.50
40–45	—	—	—	0.54
45–50	—	—	—	0.70
Calculated cumulative risk, %				
18	7.1	8.3	4.0	2.3
21	12.9	13.7	8.1	6.9
25	15.3	16.8	11.1	11.0
30	17.8	20.7	12.9	11.6
35	21.0	22.1	13.7	13.5
40	—	—	16.3	15.6
45	—	—	—	17.9
50	—	—	—	20.7
Women				
15–18	0.23	0.19	0.18	0.23
18–21	0.97	0.19	—	—
21–25	—	—	0.14	0.17
25–30	—	0.15	0.12	0.14
30–35	—	0.25	—	—
Calculated cumulative risk, %				
18	0.7	0.6	0.5	0.7
21	3.5	1.1	0.5	0.7
25	—	1.1	1.1	1.4
30	—	1.9	1.7	2.1
35	—	3.1	—	—

at the same age, except in the 4–9 years group where the figures are somewhat higher, but the differences are negligible and probably due to random variation. By comparing the groups "under 4 years" and "15 years and up" a statistically probable difference is obtained between the cumulative risks at the age of 35 (21.0 ± 2.4 and 13.5 ± 1.5). The difference is 7.5 ± 2.8 and indicates that one fifth (21%) of the 35 year-olds who were 4 or less at the time of their parents' divorce had been convicted while only one seventh (13.5 per cent)

of those who were over 15 at the corresponding time had had a similar fate. On behalf of the women no differences appear, but there seems to be a trend in the same direction.

Alcoholism in the men when seen in the same light was more riskful to those who were below 4 years old when their parents divorced, and at age 40 one third of them (cumulative risks: 33.4 per cent) had in some way been intervened against by the temperance board (see table 9). The risks then fall group by group and are lowest for those who were older than 15 when their parents divorced. Between the cumulative risks at age 40 for the youngest and the oldest group there is a statistically significant difference in the latter group's favour (21.7 ± 6.8 per cent).

Table 9. Annual risk and calculated cumulative risk in per cent of being subjected to intervention by the Temperance Board for men divided into groups according to their age at the divorce of the parents.

Age, years	Age of the sons at the divorce of the parents, years			
	Under 4	4 - 9	9 - 15	15 and over
Annual risk, %				
15-20	1.05	0.78	—	—
20-25	1.77	1.38	0.50	0.43
25-30	0.56	1.13	0.53	0.76
30-35	0.78	1.07	0.83	1.15
35-40	3.85	—	0.95	0.20
Calculated cumulative risk, %				
20	5.2	3.8	—	—
25	13.3	10.3	2.5	2.1
30	15.7	15.3	5.0	5.5
35	18.9	19.7	8.9	10.8
40	33.4	—	13.2	11.7

Social Aid among men was also most frequent in those that were youngest at the time of their parents' divorce (see table 10).

Almost two thirds of the men (60.7 per cent) who were younger than 4 years when their parents divorced had sought social aid at 40 years of age, while only just over one fourth (28.6 per cent) of those who were over 15 on the same occasion had needed to do so. The difference, 32.19 ± 8.8 , is statistically significant. The same applied to the women. At age 35 almost one third had availed themselves of social aid if they had been younger than 4 when their parents divorced,

Table 10. Annual risk and calculated cumulative risk in per cent of needing social aid for men and women when fullgrown (over 15 years) divided into groups according to their age at the divorce of the parents.

Age, years	Age of the children at the divorce of the parents, years			
	under 4	4-9	9-14	15 and over
<i>Men</i>				
	Annual risk, %			
15-20	2.00	2.37	1.12	0.61
20-25	2.76	4.78	1.09	1.55
25-30	3.29	3.90	2.70	1.16
30-35	—	2.53	1.46	1.26
35-40	10.00	1.35	0.37	2.09
	Calculated cumulative risk, %			
20	9.6	11.3	5.5	3.0
25	21.4	30.6	10.5	10.3
30	33.5	43.1	22.0	15.4
35	33.5	49.9	27.5	20.6
40	60.7	53.2	28.9	28.6
<i>Women</i>				
	Annual risk, %			
15-20	1.88	3.28	1.69	0.79
20-25	2.14	4.41	2.18	1.17
25-30	1.14	2.44	1.14	0.88
30-35	1.67	0.94	1.01	1.15
35-40	1.92	—	1.09	1.14
	Calculated cumulative risk, %			
20	9.1	15.4	8.2	3.9
25	18.4	32.5	17.8	9.4
30	22.9	40.3	22.4	13.3
35	29.1	43.1	26.2	18.2
40	35.7	—	30.1	22.7

but the same had been the case of not quite one fifth (18.2 per cent) if they were over 15 on the same occasion. These respective differences are also statistically significant.

Social Status. When the sons' occupation as grown-ups are compared to their fathers' the two youngest age groups—those under 4 and those between 4 and 9 years old when their parents divorced—exhibit the poorest results, a larger number having declined than had risen socially. The difference is statistically significant for both groups. In the two next groups—9-15 years and over 15 years—many had on the other hand lifted themselves above their fathers' social status, even if some had come down socially. So it seems that with regard to choice of occupation and vocational training those who at the time of their parents' divorce were youngest

Table 11. The social status of the sons compared with that of their fathers.
Distribution in groups according to the age of the sons at the divorce of the parents.

Fathers / Sons		Social class	
	I + II	III	Total
Under 4 years at the divorce of the parents			
I+II	21	17	38
III	19	84	103
Total	40	101	141
4-9 years at the divorce of the parents			
I+II	28	21	49
III	15	96	111
Total	43	117	160
9-15 years at the divorce of the parents			
I+II	39	18	57
III	24	83	107
Total	63	101	164
15 years and above at the divorce of the parents			
I+II	39	19	58
III	20	77	97
Total	59	96	155

also were most handicapped (see table 11), which perhaps may be attributed to economical conditions.

For all the points studied we thus find with regard to the males that those who were youngest when their parents divorced seemed to stand the greatest risk. Because the test series included too few women no similar statement would be warranted with regard to them. To some extent, therefore, the result has gone against what one would have expected and showed that the old are at lesser disadvantage than the young, by whom a divorce between the parents theoretically should pass without leaving much trace. It is conceivable that the youngest group might have inherited more undesirable traits than the others. A study of the frequency of criminal parents in the different groups showed, however, that they were distributed fairly evenly, and there was no difference between the two extreme groups in this respect. Nevertheless it is possible that people who divorce early when the children are small might find it more difficult to adapt themselves than those who divorce late after many years of married life. Such lack of adaptability might spring from hereditary traits that might be transmitted to the children. The less favourable

environment which mostly follows the parents' divorce unfailingly must on the other hand be more hard on the children the younger they are at the time of the divorce. No conclusions can therefore be drawn concerning the effect of the divorce as such or with regard to the possible role of the hereditary background.

Custodianship.

Another question which comes to mind is this: Does it affect the outlook for the child into whose custody it is given—the father's or the mother's?

When the court decides which parent is to have custody of the child or children the usual thing in Sweden is to award them to the mother. If the parents have made an agreement or if insanity or some other reason makes the mother an unsuitable custodian, they are as a rule awarded to the father. Whenever both parents are considered unfit a guardian is appointed.

The 58 young people who were of age when their parents divorced having been deducted, there remained 1270 minors. Of these 540 boys and 546 girls were awarded the mother while 104 boys and 80 girls were awarded the father. Thus more than four fifths came into the mother's custody and less than one fifth into the father's. Three boys were in the custody of relatives, and for one boy and one girl the custodianship could not be ascertained. In most cases all the children in one family were awarded to one or other of the parents, but the children in 40 families were "shared" between the parents.

If the results of the prognosis are considered in the light of the custodianship, it will be seen with regard to *measures taken by the child welfare board* that many more boys in maternal than in paternal custodianship were "warned". The difference is not significant, however, and with regard to the more severe form of intervention, detention for reform schooling, no difference could be established whether the father or the mother had custody of the child.

The reverse turned out to be true of daughters. With regard to "warnings" there was no difference between the custodians, whereas reform schooling much oftener was resorted to if the girls were in the fathers' charge than in the mothers'. This difference is not significant either. In both cases we probably have to do with an effect of random variation; and, as stated previously, a correct evaluation of the child welfare board's interventions would have required further subdivision which the material did not permit.

With respect to *criminality* it would seem that children who grew up in the mother's custody ran the greatest risk. This applies to both boys and girls irrespective of their age at the time of their parents' divorce. While the cumulative risk for males brought up by the mother was 22 per cent at age 45, it was only 10.5 per cent if they had been with the father (see table 12). The difference is

Table 12. Calculated cumulative risk in per cent of committing crimes, of being subjected to intervention from the Temperance Board and of needing social aid for men with divorced parents, divided into groups according to their being in the custody of the father, respectively of the mother, after the divorce.

Age, years	Criminality		Age, years	Alcoholism		Social aid	
	Father custodian	Mother custodian		Father custodian	Mother custodian	Father custodian	Mother custodian
18	5.0	5.7	20	3.0	2.2	6.9	7.8
21	6.0	11.1	25	7.2	7.1	14.1	19.7
25	8.1	14.5	30	7.2	11.3	25.7	29.5
30	10.5	16.6	35	12.2	15.0	28.9	35.2
35	10.5	18.4	40	12.2	17.7	31.6	40.3
40	10.5	20.9	45	18.8	19.0	31.6	42.3
45	10.5	22.0					

11.5 ± 2.16 and is evidently significant. With regard to the females it will be seen that only one of the 80 that were brought up by the father had her name in the penal register—the same frequency as in the population at large: 1.1 per cent—whereas those brought up by the mother showed double the percentage (2.2%). The same trend as before, i.e. greater risks for those who were younger at the time of the divorce, also emerges here in respect of the children who were brought up by the mother. When the father was made custodian, on the other hand, no such trend could be observed.

Alcoholism. Concerning the risk of running foul of the temperance board there was on the other hand no difference whether the subject had as a child been with the father or with the mother. At age 45 the cumulative risk was 18.8 and 19.0 per cent respectively. Here too, however, it was observed for those brought up by the mother that the risk was greater the younger the child at the time of the divorce. Owing to the small number of children awarded to the father, it cannot be determined if the same would have been the case of these children.

Social Aid. The risk of having to apply for social aid during adulthood was once again greater for those who were brought up

by the mother. At age 40 the cumulative risk for men was 40.3 and 31.6, respectively, if they had been in the mother's or in the father's custody (cf. table 12). The statistically probable difference is 8.75 ± 3.14 .

Yet once more the need was greater for the youngest age groups and particularly prominent for those in the mother's care.

The females' need for social aid was the same whether they had been brought up by the father or by the mother and there was no appreciable difference between the age groups. The women's need for social aid is difficult to evaluate, however, as often a marriage intervened which might have a positive or a negative effect.

In the matter of the sons' *social status* when grown up it will be seen that among those who had been in the father's custody about as many belonged to social classes I and II as belonged to social class III, a distribution fully equivalent to that displayed by their fathers at the time of the divorce. If the fathers belonged to social class III the same number of sons lifted themselves to class I and II as came down from the higher classes to which their fathers belonged. Accordingly, as many sons who had been brought up by their fathers improved their social status as went down one step, in either case 22 per cent.

Concerning those men who had grown up in their mothers' care it was found that rather more had come down socially than had worked themselves up. The respective figures are 40.0 and 19.2 per cent, and the statistically significant difference 20.8 ± 4.7 per cent.

In other words, whether they had been brought up by the father or by the mother about the same number of sons improved their social position, but a larger number of the boys that had grown up in their mothers' care had to remain content with a lower social status than those who stayed with the father. The difference, 18.3 ± 4.7 , is statistically significant.

Thus, interventions by the temperance board excepted, we find less favourable figures for children cared for by mothers than for those awarded the father. That is not to say, however, that the father would be preferable as custodian. It should be remembered that even if the father as a rule has economical resources he is seldom in a position to bring the children up directly himself. He must enlist female help, either with relatives or by giving the children a step-mother.

The courts are most liable to let the mother keep the children, the oftener the younger they are. That the final results in favour of the father can be attributed to this fact—as already proved, those belonging to the youngest year groups at the time of the divorce, are worst situated—cannot, however, be demonstrated here.

Nor can the hereditary background—manifested as criminality or alcoholism in either of the parents—be shown to differ according to the custodianship.

And a final question: Has the social status of the parents any significance for the development of divorcee's children as grown-ups?

It is very likely that families from social classes I and II, having greater economical resources, offer better opportunities of protecting the children from being involved in the parents' squabbles and better external environments than families in which poverty is combined with parental irresponsibility and in which scenes of alcoholic excesses and brutality towards the wife have been everyday occurrences. That the parental social status on the whole is not without importance for the social behaviour of the children when they have grown up probably cannot be denied. And there is no reason why all this should not apply to children even if their parents have divorced each other. In order to study the matter we have divided the test series into two groups in accordance with the father's occupation. In so doing it was necessary to combine social classes I and II because of the small number of subjects.

With regard to *measures taken by the child welfare board* it is highly probable that there will be a difference in frequency between children whose fathers come from different social classes. For parents from the two upper classes have more resources to arrange for their maladjusted children without turning to the child welfare board, and this must make a difference for divorcees' children as well. The fact revealed by the investigation that a greater number of both boys and girls were given "warnings" or sent to reform schools if their fathers belonged to social class III than if they came from either of the two upper classes is therefore by no means surprising, and not at all something peculiar to divorcees' children.

Coming now to the figures for *criminality* in the men, we unequivocally find that the risks are far greater for those whose fathers came from social class III than for those whose fathers came from the other classes (cf. table 13). The cumulative risks at 40 years of age for the former group is 23 per cent and for the latter 10.1

per cent, the difference being 12.9 ± 1.8 per cent and statistically significant.

Table 13. Calculated cumulative risk in per cent of committing crimes, of being subjected to intervention from the Temperance Board and of needing social aid for men with divorced parents distributed according to the social status of the father at the divorce.

Age, years	Criminality			Alcoholism			Social aid	
	Social class of the father		Age, years	Social class of the father		Social class of the father	I + II	III
	I + II	III		I + II	III		I + II	III
18	2.8	6.6	20	0.5	3.2	2.8	9.5	
21	5.1	12.8	25	1.1	9.9	6.3	23.7	
25	6.6	16.7	30	2.3	14.3	11.6	35.5	
30	7.2	19.4	35	6.6	18.7	15.7	40.9	
35	8.8	20.8	40	6.6	21.9	18.3	47.3	
40	10.1	23.0	45	6.6	24.8	18.3	50.2	
45	10.1	25.2						

Alcoholism. The figures for the various categories reveal that the risk of incurring an intervention from the Temperance Board is considerably higher for those whose fathers came from social class III than for those whose fathers came from the other classes (see table 13). The cumulative risk at 35 years of age for men is 6.6 per cent if their fathers came from social classes I and II—and thus approximately equal to Dahlberg's normal figure: 6.1 per cent—but if the fathers came from social class III the corresponding cumulative risk is 18.7 per cent, i.e. almost 3 times higher. The difference is 12.1 ± 1.45 per cent and is statistically significant.

At this point it might be profitable to look into another matter: Which of the two sexes runs the greatest risk of being harmed by the parents' divorce, the boys or the girls? It is impossible, however, to say anything definite in the matter from the data available.

The importance of genetic factors in this connection has also been studied. Thus the frequency of criminality among children whose father or mother had been convicted of a felony was compared with the frequency among those whose parents had not been sentenced. No difference appeared, but the frequency was about the same in both groups, for men as well as for women.

But the figures sing a different tune with regard to alcoholism. When sons of fathers who abused alcohol are compared with sons whose fathers did not, a clear difference emerges to the disadvantage of the former group. At an age of 40 years the cumulative risk for the first group was 30.3 ± 2.6 per cent and for the second 14.4 ± 1.1 per cent. Being 15.9 ± 2.9 the difference is statistically significant.

Judging by the figures it would thus seem that inherited traits weigh more heavily in the production of alcoholism than of criminal tendencies.

But divorcees' children obviously do not inherit only such traits as manifest themselves in the form of alcoholism or criminality. To them might also be transmitted genes which give rise to an inadequate ability to adapt themselves to the requirements of society. From such a point of view it is not surprising that the prognosis for divorcees' children is bleaker than for other children. However, it is impossible to escape the fact that the peculiar environment in which divorcees' children grow up must contribute to the poor prognosis: the divorce, as noted, often forces the mother to take employment and her children will be less well looked after. A divorce that comes early when the children are small must obviously

Prognosis for children of divorcees.

Prognosis	Boys	Girls
Measure by Child Welfare Board	13.4 %	2.7 %
Detention for reform schooling	6.6 %	2.1 %
Detention for reform schooling, normal figures.	1.5 %	0.97 %
Criminality, cumulative risk up to 50 years	22.9 %	2.5 %
Criminality, normal figures according to Dahlberg, cumulative risk up to 50 years	9.9 %	1.1 %
Measure from Temperance Board, cumulative risk up to 40 years	17.0 %	
Measure from Temperance Board, normal figures according to Dahlberg, cumulative risk up to 40 years	6.1 %	
Sentence for drunkenness, cumulative risk up to 35 years	14.3 %	
Sentence for drunkenness, normal figures according to Dahlberg, cumulative risk up to 35 years	7.3 %	
Social aid, cumulative risk up to 45 years	40 %	34 %

be particularly undesirable. It is nowadays held that early impressions mark a person for life. It is likely, moreover, that parents who have an early divorce, when the children are still small, ought to have a more marked inability to adapt themselves and therefore transmit especially undesirable genes to their children. In this text the terms traits and genes are used without reference to identifiable qualities which produce psychopathy and the like.

Summary.

On the basis of this investigation it is probably justified to say that children whose parents have divorced have a more dismal prognosis than children in general. Even if the divorce itself, its effects on the childrens' mentality, and the subsequent change of environment have a great responsibility in this matter, the hereditary background is obviously jointly responsible if not decisive. It has appeared that the parents more often are criminals or alcoholics than people in general; and it is justifiable to assume that at bottom the divorce is caused by a variety of traits that make it impossible for the parties to live together. When a predisposition to such traits is transmitted from both parents it now is not surprising that the children will find it still more difficult to get on with other people and to manoeuvre in the maze of society. However, it is impossible to gather from this investigation whether one or the other of these factors is decisive. At present the author is collecting data which are more suited to compare the roles of divorce and heredity respectively.

The result of the investigation of the prognosis for sons and daughters of divorced parents is given in the table on page 94 which also gives figures for the normal population.

Résumé.

En vertu de cette investigation on peut probablement dire que les enfants dont les parents ont divorcé ont un prognostic moins bon qu'ont les enfants en général. Même si le divorce seul, ses effets sur la mentalité des enfants ainsi que le changement de milieu en a une grande responsabilité, il est évident que la disposition héréditaire doit jouer aussi un rôle. Il s'est montré que les parents sont criminels ou alcooliques plus souvent que les gens en général et il y a lieu de supposer qu'au fond le divorce dépend d'une multitude de traits qui font échouer la vie commune des époux. Quand une telle disposition

est transmise héréditairement des deux parents, il n'est pas étonnant que les enfants aient de la peine à s'accorder avec d'autres personnes et de se débrouiller dans la société. Cependant, sur la base de l'investigation présente il n'est pas possible de dire lequel de ces facteurs soit décisif. A présent l'auteur est en train de recueillir des données qui peut-être pourront mieux élucider les rôles respectifs du divorce et de l'hérédité.

Le résultat de cette analyse prognostique pour les enfants des parents divorcés se trouve dans le tableau page 94. Aussi des chiffres pour la population normale sont donnés.

Zusammenfassung.

Auf Grund dieser Untersuchung ist es wahrscheinlich gerechtfertigt zu sagen, daß Kinder, deren Eltern geschieden sind, eine dunklere Prognose haben als Kinder im allgemeinen. Selbst wenn die Scheidung an sich ihre Wirkungen auf die Mentalität der Kinder und der darauffolgende Wechsel der Umgebung eine große Verantwortung in dieser Angelegenheit haben, so ist der erbliche Hintergrund auffallend mitverantwortlich, wenn nicht entscheidend. Es hat sich gezeigt, daß die Eltern öfter Verbrecher oder Alkoholisten sind als die Menschen im allgemeinen; man kann wohl rechtmäßig annehmen, daß die Scheidung im Grunde durch eine Verschiedenheit von Charakterzügen verursacht wird, welche es den beiden Parteien unmöglich macht, zusammenzuleben. Wenn die Veranlagung zu solchen Charakterzügen von beiden Eltern übertragen wird, kann es nicht überraschen, daß die Kinder es noch schwerer haben werden mit anderen Leuten auszukommen und in dem Labyrinth der Gesellschaft zu manövrieren. Es ist jedoch unmöglich aus dieser Untersuchung zu schließen, ob der eine oder der andere von diesen Faktoren entscheidend ist. Zur Zeit sammelt der Verfasser Angaben, welche zum gegenseitigen Vergleich der Rollen von Scheidung und Vererbung besser geeignet sind.

In der Tabelle auf Seite 94 wird das Ergebnis der Untersuchung der Prognose für Söhne und Töchter geschiedener Eltern aufgezeigt; desgleichen werden Ziffern für Normalbevölkerung angegeben.



Soeben erschien:

LEHRBUCH DER GERICHTLICHEN MEDIZIN

von

J. Dettling
Bern

S. Schönberg
Basel

F. Schwarz
Zürich

VIII + 552 Seiten und 134 Abbildungen sFr. 54.10

Aus dem Vorwort:

Beim Ende des letzten Weltkrieges waren praktisch sämtliche Lehrbücher dieses Faches innerhalb des deutschen Sprachgebietes verschwunden. Die Nachfrage sowohl bei Studierenden als auch bei Ärzten und Juristen war sehr groß. Daher haben die Autoren damals den Entschluß gefaßt, nach sorgfältiger Vorarbeit ein neuzeitliches Lehrbuch herauszubringen. Aber nicht nur der Mangel an guten klassischen Werken erforderte die Neuschaffung eines Lehrmittels, sondern mehr noch die großen Fortschritte, welche die Gerichtsmedizin in den letzten Dezennien zu verzeichnen hatte, und das ständige Steigen der Anforderungen, welche in mannigfacher Hinsicht an sie gestellt werden. Es sei nur an die gewaltige Zunahme des Verkehrs und damit auch der Unfälle gedacht, die zahlreichen Maßnahmen und gesetzlichen Bestimmungen zur Verringerung der Gefahren, die Rolle des Alkohols bei der Unfallsauflösung. Auch die enorme Entwicklung der chemischen Wissenschaft und Industrie hat den Gerichtsmediziner vor immer größere Aufgaben gestellt. Es sei auch erinnert an die Bedeutung der Blutuntersuchung zur Entscheidung in Vaterschaftsfragen, Blutgruppen, Rhesusfaktor, eine Forschung, die durch ihre Ergebnisse heute in der Rechtsprechung allgemein fest verankert ist.

Aus dem Inhalt:

Allgemeine gerichtliche Medizin – Die forensisch-medizinischen Wirkungsmittel – Die Basis der ärztlichen Wirksamkeit: Stellung als diplomierter Arzt; die Gesetze – Das ärztliche Geheimnis; Berufsgeheimnis; Berufspflicht; Falsches ärztliches Zeugnis; Ärztliche Anzeigepflicht – Allgemeine Grundsätze für die Erstellung von ärztlichen Zeugnissen und Gutachten – Die Lehre vom Gutachten – Der außerordentliche Todesfall – Die Leichenerscheinungen – Der plötzliche Tod aus natürlicher Ursache – Der Selbstmord in der gerichtlichen Medizin.

Spezielle gerichtliche Medizin – Allgemeine Gesichtspunkte für die Beurteilung von Körperverletzungen und Übersicht der medizinischen Aufgaben – Die Schnittverletzungen – Die Stichverletzungen – Hiebwunden bei schneidenden Objekten – Die stumpfe Gewalt – Die Quetschung – Die Gewebszerreißung – Schartenspuren – Der Automobilunfall – Motorrad- und Velounfälle – Todesfälle auf dem Eisenbahntrasse – Flugzeugunfälle – Absturz – Die Schußverletzungen – Tod und Gesundheitsschädigung durch abnorm hohen und abnorm tiefen Luftdruck – Schädigung durch abnorm hohe und abnorm tiefe Temperaturen – Der elektrische Unfall – Hungerkrankheiten und Hungertod – Der Tod durch gewaltsame Erstickung – Die Kindestötung – Fragen der Schwangerschaft, Geburt und Fruchtabtreibung – Gerichtlichmedizinische Untersuchungen bei Sexualdelikten.

Gerichtlich-medizinische Spurenkunde einschließlich Blutgruppen.

Die Toxikologie im Rahmen der gerichtlichen Medizin.



S. K A R G E R

B A S E L N E W Y O R K

*Neuerscheinungen***Atlas der
Elektrokardiographie**

von O. RITTER und V. FATTORUSSO

256 Seiten, 219 Abb. sFr. 36.40

Es ist den Autoren gelungen, eine moderne Elektrokardiographie zu schreiben, welche nicht nur alle Theorien, sondern auch die klinischen Gesichtspunkte berücksichtigt. Das Werk wendet sich daher nicht nur an den Spezialisten, sondern an alle Ärzte, die mit Fragen der Elektrokardiographie zu tun haben.

Progress in AllergyFortschritte der Allergielehre
Vol. IIIEdited by / Herausgegeben von
P. KALLÓS572 p., 89 fig., 64 tables, 1 colored plate, 1952.
sFr. 68.65

The reviews on "Allergy in Children" by M. M. Peshkin, and those on "Asthma" by L. Unger, A. H. Rowe, D. Harley and by F. Wyss and W. Hadorn give important and concise information concerning these fields. J. Harkavy summarizes his own experience in the field of "Cardio-Vascular Allergy". E. Rothlin and R. Bircher as well as A. Stoll give a review on ergot-alkaloïds, while P. Kallós and L. Kallós-Deffner discuss the therapeutical use of Gynergen. The Articles by E. A. Brown and J. F. Ackroyd deal with problems of "DrugAllergy" from clinical and experimental points of view.

Die biogenen Amine

und ihre Bedeutung für die Physiologie und Pathologie des pflanzlichen und tierischen Stoffwechsels

von Dr. phil. et med. h. c. M. GUGGENHEIM,
Basel. 4., neubearbeitete Auflage.
XVI und 619 Seiten. sFr. 78.-

Die vorliegende Neubearbeitung gibt einen Überblick über Vorkommen, Biogenese, physiologische Bedeutung, biochemisches und pharmakologisches Verhalten, Eigenschaft und Salze, Nachweis und Bestimmung der biogenen Amine.

**Fortschritte der
Tuberkulose-Forschung**Advances in Tuberculosis Research
Progrès de l'Exploration de la Tuberculoseherausgegeben von H. BIRKHÄUSER, Basel
und H. BLOCH, New YorkVol.I/II(1948/49): IV und 446 Seiten, 4 Abb.
sFr. 52.-Vol.III(1950): IV und 307 Seiten, 13 Abb.
sFr. 39.60

Vol. IV (1951): 308 Seiten, 59 Fig., sFr. 43.70

Die Mitarbeiter der «Fortschritte» erstreben eine möglichst vollständige Erfassung der Literatur; besonders aber wollen sie eine Gesamtdarstellung der Entwicklung ihres Spezialgebietes geben. Die Abhandlungen dienen dem Nichtspezialisten als Einführung in die Problemstellung, dem Spezialisten zur kritischen Wertung des neuesten Standes der Forschung.

**Lehrbuch der
Gerichtlichen Medizin**von Prof. Dr. J. DETTLING, Bern;
Prof. Dr. S. SCHÖNBERG, Basel, und
Prof. Dr. F. SCHWARZ, Zürich

552 Seiten mit 133 Abb., sFr. 54.10

Die Medizinische Welt (Nr. 31/32, 1951): «Das Buch eignet sich insbesondere auch zum Nachschlagen für den Arzt, Amtsarzt, gerichtliche Medizin treibenden Pathologen, Juristen und Kriminalisten und muß warm empfohlen werden.»

**Lehrbuch der
Haut- und Geschlechts-
krankheiten**

von Prof. Dr. W. LUTZ, Basel

X und 658 Seiten mit 375 Abb. sFr. 66.55

Durch die zahlreichen, mit Sorgfalt ausgewählten Abbildungen wird das Buch gleichzeitig auch zu einem Atlas für die tägliche Praxis.